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ARCHIVES OF SURGERY

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MÉNIÈRE'S DISEASE

ITS DIAGNOSIS AND A METHOD OF TREATMENT *

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The purpose of this communication is to present the results of an operation which I believe will permanently cure the symptoms of Ménière's disease. Briefly stated, the treatment is section of the auditory nerve intracranially. It is attended with almost no risk of life, and since there is always subtotal deafness on the affected side before the operation, section of the nerve adds little of practical importance to the deafness. Other symptoms do not result when the auditory nerve is severed.

In its usual form, Ménière's disease has a well defined and well recognized symptom complex. The patient is suddenly seized with a violent attack of dizziness, at once associated with nausea, vomiting and unilateral tinnitus referred to an ear which is progressively growing deafer. These attacks are repeated from time to time, usually with increasing frequency. The patients are well between the attacks, though eventually they may recur so frequently as to be almost continuous. At such times, as for example in case 1, the patient for weeks may not be able to take food or to retain it when it is taken. The attacks are of such violence and come on with such suddenness that the patient lives in terror of their reappearance. They last from a few hours to several weeks. The symptoms have been known and described for more than a century, but it was Ménière who first suspected their aural origin. Whether or not his impression proves to be correct, he at least rescued a clinical entity from a hopeless confusion of symptoms which had not implied any pathologic significance. At the time of Ménière's publication, the symptoms of this condition, and of epilepsy also, were considered evidence of cerebral congestion or apoplexy. Indeed, the sudden nature of the attacks of Ménière's disease might well appear at first glance to be apoplectiform.

It is necessary to understand the beliefs current at that time in order not to be misled by the title of Ménière's paper "Maladies de l'oreille interne offrant les symptomes de la congestion cérebrale apoplectiforme." There is even yet the erroneous impression (perhaps attributable to the

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title) that Ménière thought this condition was due to cerebral hemorrhage or to hemorrhage into the semicircular canals. He made it clear that the symptoms are considered by all authorities to be due to cerebral congestion, but he repeatedly emphasized his belief that they are surely due to a disease of the internal ear. Elsewhere he said "The signs of cerebral congestion, so called, are not too well established; and—the signs of cerebral congestion and apoplexy will have to be revised." Ménière referred to the work of Itard (published in 1825) in his paper. At times, Itard is unjustly given credit for the discovery of Ménière's syndrome, but, according to Ménière he assembled cases of epilepsy, hysteria and other conditions with those of aural vertigo and classified all of them as "cerebral apoplexy." It is interesting that at the meeting of the Academy of Medicine of Paris when Claude Bernard was almost unanimously elected to be a member, the subject of cerebral congestion was discussed at length, and Ménière's work was cited as argument in opposition. It was the sense of the meeting that since "there was so much contradiction, confusion and exaggeration on this subject it was better to separate epilepsy and confine the attention to true cerebral congestion." Ménière maintained that there could not be any possible relationship between epilepsy and aural vertigo, because epileptic persons do not have unilateral deafness, and patients with aural vertigo do not lose consciousness nor have the mental stigmas so common to persons with epilepsy.

Ménière's belief in the site of the lesion in the disease which has since borne his name was largely due to the well known experiments on extirpation of the semicircular canals by Flourens (1842). Ménière also reported gross observations made at necropsy in one case with acute symptoms, which persisted until death occurred five weeks later. The semicircular canals were found to contain "serosanguineous lymph." The brain did not show any abnormality. The observations made at necropsy by Ménière are obviously those of some acute aural condition and are not those of Ménière's disease which is now known as a chronic disease. The necropsy report of Ménière cannot be used to support his clinical syndrome, unless it be to show that such symptoms may be present in an acute form and may be referable to a lesion of the inner ear and not to the brain.

There is, in fact, no available material to indicate the character of the underlying lesion of Ménière's disease. A few cases have been reported, but all are complicated by other lesions. Politzer's patient had a tumor of the brain, Voltolini's had meningitis; Gruber's patient died of typhus and had blood tinged lymph in the semicircular canals.

Evidence that Ménière's disease is due to a lesion of the inner ear is based almost entirely on the presence of deafness. In the absence of proof, it is permissible to doubt that the pathologic process is actually in

the semicircular canals. In fact, by analogy with other conditions, it is easier to believe that the nerve itself and not the end-organ may be primarily involved. It is evident from histories of Ménière's disease that infections of the middle ear have no etiologic relationship. Ménière early called attention to the absence of otitis media and of hereditary factors in the explanation of an underlying case.

TREATMENT IN MÉNIÈRE'S DISEASE

Charcot (1874), who frequently demonstrated cases of Ménière's disease at his clinics, admitted the utter hopelessness of all forms of treatment then in vogue. He noted that when deafness became complete the disease stopped spontaneously. This observation prompted him to wonder if surgical intervention might not at some future time offer the solution by dividing the auditory nerve. But this suggestion was made when antiseptic surgery was just beginning and several years before the dawn of brain surgery. With one exception the attempts at surgical treatment have been directed toward the semicircular canals.

Frazier (1912), following a suggestion by Mills (1908), divided the auditory nerve intracranially in a single patient and precisely as in the operation here described, but the dizziness was not relieved. A study of Frazier's case leads one to doubt that the diagnosis of Ménière's disease was correct; the characteristic "attacks" of dizziness, nausea and vomiting were not present. Rather there was a chronic state in which dizziness could be induced by postural change. The patient also was unable to lie in comfort on the contralateral side. These symptoms would seem to indicate involvement of the cerebello —brain stem— vestibular paths by a neoplasm or inflammatory process. Furthermore, there was the history of influenza quickly followed by deafness—an etiologic relationship not recognized in Ménière's disease.

Section of the eighth nerve for persistent tinnitus was then tried by Frazier (1913), but the ultimate success is not known. He stated that "the intense roaring sound" had disappeared at the time of the patient's discharge from the hospital. The results in the cases reported in this paper make one wonder how far section of the eighth nerve can control noises referred to the ear.

Other surgical attempts at treatment in cases of Ménière's disease have been directed toward direct attacks on the inner ear after chiseling through the mastoid. Houtant trephines the labyrinth; Portmann incises the saccus endolymphaticus. Aboulker exposes the dura behind the mastoid and expects to "decompress" the auditory nerve "either with or without opening the dura." The assumption of Aboulker that an increase of pressure of the cerebrospinal fluid in the posterior cranial fossa was responsible for Ménière's disease was due to Babinski's claim that lumbar punctures relieved the symptoms. At a meeting of the Inter-

national Congress of Otology in 1922, this theory was supported by Nylen (Stockholm) and Quix (Utrecht). In refutation of this assumption it is only necessary to say that Ménière's disease is never seen when the intracranial pressure is really increased as in tumors of the posterior cranial fossa. An increase of pressure around one auditory nerve, aside from being pure conjecture, is impossible because the cisterna lateralis which surrounds the auditory nerve is freely open in three directions. In none of my operative series was there the slightest evidence of increased intracranial pressure, either local or general.

REPORT OF CASES

CASE 1.—*History.*—A well nourished man, aged 53, was referred by Dr. G. H. Barksdale, of Charleston, W. Va., in September, 1924, because of vertigo, nausea, vomiting and buzzing in the left ear. The symptom of the onset was a unilateral buzzing in the ear which began two and one-half years previously. Impairment in hearing was noticed at the same time and had progressed gradually. Two months before examination, a headache began in the morning; later in the day, the patient became very dizzy. He went to bed, but the vertigo continued throughout the day. There were severe nausea and persistent vomiting. Everything seemed to move to the left. He said that he believed he would have fallen to the left if he had not gone to bed. Other attacks occurred but they did not last longer than a day. Three days before admission he had the most severe attack which wakened him out of a sound sleep. He saw objects moving to the left, and was greatly nauseated for five hours. Numerous attempts to vomit were ineffectual. A friend who saw him in this attack said that the temperature was 97 F. and the pulse rate, 60. For the past few months he had had a little headache over the occiput. The headaches were never severe, but were much worse during the attacks. Ringing in the left ear had been present since the onset of the condition. It was more intense during an attack. There had never been infection of the middle ear or mastoid.

Neurologic and Physical Examination.—With the exception of a questionable positive Romberg test (with tendency to fall to the left) and the disturbance referable to the eighth nerve, the results of the neurologic examination were negative. The patient had a bradycardia (60) during an attack. The Wassermann reaction of blood was negative. According to the tests the hearing in the left ear was moderately impaired; a watch tick was not audible. *C* 64 was not heard in the left ear; *C* 128 was heard only one fourth of normal time. *C* 1024 and *C* 2048 were not greatly changed. Bone conduction was greater than air conduction.

Vestibular tests showed that spontaneous nystagmus was not present. There was a normal response to caloric stimulation of both ears, but the left ear (affected side) was more quickly induced than the right.

Operation.—The left eighth nerve was divided intracranially under local anesthesia. The patient did not experience any sensation when the auditory nerve was dissected and divided. The postoperative course was uneventful.

Three and a half years after the operation, the patient was in perfect health and has been actively at work for the past three years. He has never had the slightest suggestion of an attack since the operation.

CASE 2.—*History.*—A well nourished woman, aged 54, was referred to me by Dr. Julian Chisolm, on Nov. 10, 1924. For three or four years the patient had had occasional attacks of dizziness and vomiting. At first they were not severe. One year before admission, she had a severe attack associated with ringing in the left ear and some impairment of hearing in the left ear. She was confined to bed for four days and found that she was much more comfortable when lying on the left side. Two months later, an even more severe attack prostrated her. It was like the preceding one, except that there was pain in the left occipital region and occasional tingling and numbness of both hands. There were also zigzag flashes of light. For the past three months before consulting me there had been a constant tinnitus in the left ear, some unsteadiness of gait and a constant dull ache in the region of the left mastoid. There was no history of otitis media.

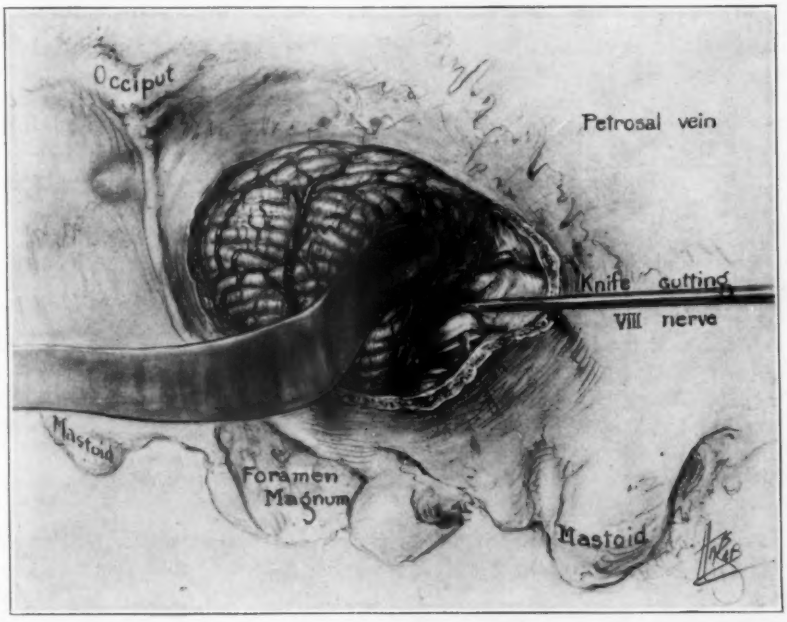

Fig. 1.—Operative approach to region of the eighth nerve. The nerve can be divided either with a blade at right angles to a long shaft, or with a small pair of scissors.

Examination.—The results of the physical examination and the Wassermann reaction of blood were negative. The results of the neurologic examination were negative, except for changes referable to the auditory nerve.

The hearing test revealed total deafness in the left ear; the right ear was normal.

The caloric test did not show any response to irrigations of the left ear; the right was normal.

Operation.—The left auditory nerve was sectioned intracranially on Nov. 12, 1924, under ether anesthesia. The patient was discharged from the hospital ten days later.

She has not had any attacks since operation. She complains of a drawing, tight feeling in the occipital region at the operative field, and zigzag flashes in

the eyes; this sensation has been present for more than twenty-five years. Sometimes there is a little dizziness with the flashes of light (migraine?).

CASE 3.—*History.*—An undernourished woman, aged 55, was referred by Dr. Barker on Jan. 11, 1927, because of Ménière's disease. She was exhausted from prolonged vomiting. Her skin was sallow and pale. Eighteen months before, she was suddenly seized with dizziness, nausea and vomiting. The attack lasted all night. The vomiting was described as projectile. She did not have any fever, chills or pain. She had never been well since the onset of the condition. Six weeks later, she had another attack precisely like the first.

The interval between attacks gradually lessened, until the patient was admitted to the hospital. Each attack lasted one or two days. Only hypodermic injections of morphine gave any relief. Six months before coming to the hospital, she had a prolonged attack during which she did not take food for five weeks. A diagnosis of gallstones had been made in her home town, and the gallbladder had been drained. Gallstones had not been present. The appendix had also been removed at the operation. Her attacks of dizziness, nausea and vomiting persisted as before, though possibly with some moderation. Five weeks before admission her most severe attack began and still persisted. Since the attack began, she had had scarcely any food. She said she felt as if she were going round, first one way, then another. The attacks were not brought on by change of position, but during an attack she could not lie on either side. Sudden movements of the head then intensified the attack. There had been a gradually progressive loss of hearing in the right ear since the onset of the present illness. At the time of consultation, she could hear but little on the right side. There was also a continual ringing in this ear which became much worse during attacks. Recently, there had been some ringing in the left ear. An occasional slight pain was experienced in the right ear. There had never been an infection of either ear or mastoid. Nothing in her past history appeared to have any bearing on her condition. An attack of rheumatism, localized to the right hip, was noted thirty-five years before. During the menopause, two or three year previously, cyanosis of the fingers had been noted. She had always been nervous. Since her trouble began (eighteen months before), she lost 26 pounds (11.8 Kg.).

Examination.—The patient was in Dr. Barker's medical service for one month. There were few days during this time when the dizziness, nausea and vomiting were not present. The vomiting was often projectile. During most of this time she could retain nothing by mouth and was given fluids by rectal, subcutaneous and intravenous methods.

Except for the disturbed functions of the eighth nerve, the results of the neurologic examinations were entirely negative.

Examinations of auditory and vestibular functions were made by Dr. Baylor. All tuning forks were heard on the left side within normal limits. On the right, there was marked shortening with all forks. Vibrations of tuning fork *C* 128 were barely heard to *C* 2048.

Irrigation of the right ear produced a rotary nystagmus to the left, but no subjective dizziness. Irrigation of the left ear caused a rotary nystagmus to the right, but no subjective vertigo.

The audiometer test showed loss of hearing in the right ear and of some of the high tones in the left ear. The vestibular function was not abnormal.

The Wassermann reaction of the blood was negative. The blood pressure was 110 systolic and 70 diastolic.

Operation.—On Jan. 11, 1927, intracranial division of the right auditory nerve was performed under local anesthesia. The patient was not conscious of any

sensation either when the auditory nerve was being liberated or when it was being divided. The postoperative course was uneventful.

Fourteen months after the operation, there had not been the slightest evidence of an attack. She had regained her original weight and more and was well.

CASE 4.—*History.*—A large robust man, aged 47, was referred in April, 1927, by Dr. F. C. Schreiber of Washington. One year before, the patient had felt dizzy when he awoke and raised his head. He vomited, but was not nauseated. He was bedfast for three days. He felt dizzy every time he moved his head and vomited nearly as often. It was three weeks before the dizziness entirely disappeared. Four months later, he had another attack exactly like the first but this lasted only a week. Seven months before, tinnitus and deafness developed in the left ear; both appeared suddenly during an attack of dizziness and vomiting. The attacks of dizziness were not associated with moving objects. Headaches were not present. The patient reeled and staggered when walking during an

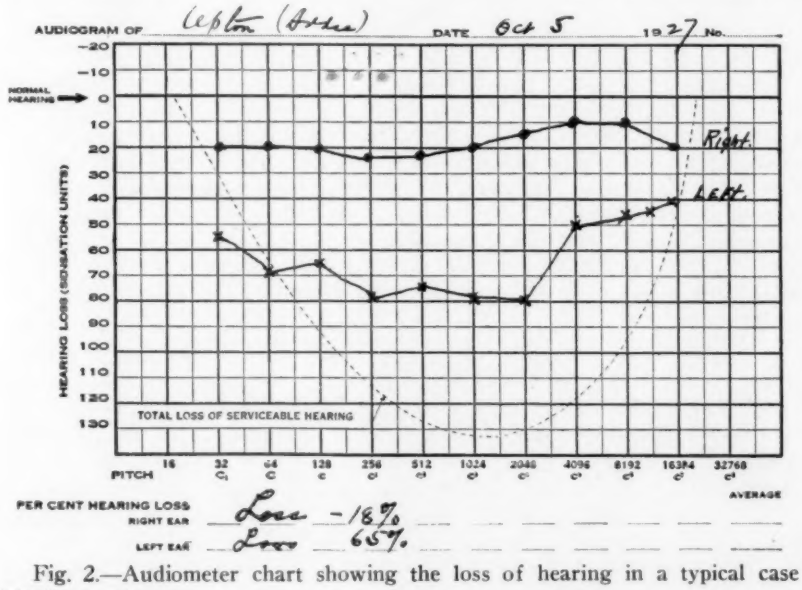

Fig. 2.—Audiometer chart showing the loss of hearing in a typical case of Ménière's disease.

attack. During the past month, there had been no additional attacks, but the patient was always dizzy. He complained of poor memory during the recent attacks.

Physical and Neurologic Examinations.—A positive Babinski sign and ankle clonus were present on the left side. The only other positive symptoms were the disturbed function of the eighth nerve. Unfortunately, the audiometer and caloric records of the functions of the vestibular and auditory divisions of this nerve were lost. It was recorded, however, that air conduction was less than bone conduction in the left ear, and that hearing was lost for the high tones and diminished for the low tones.

The blood pressure was 125 systolic and 85 diastolic. The Wassermann reaction of blood was negative.

The unilateral Babinski sign and ankle clonus made the diagnosis of Ménière's disease rather uncertain.

Operation.—In March, 1927, the left acoustic nerve was divided intracranially under local anesthesia. Thorough exploration of the cerebellopontile angle did not disclose a tumor. The entire intracranial course of the eighth nerve was in full view. Later, the symptoms did not clear up, and it was found by caloric tests that vestibular function was still present, though there was total deafness. Apparently, in an effort to avoid the facial nerve, the vestibular division of the nerve had not been divided. Two weeks later, the wound was reopened and this branch sectioned. The facial nerve was also injured despite caution. This was the only injury to the facial nerve in the series. It was later corrected by a spinofacial anastomosis.

The patient had not had attacks of dizziness and vomiting one year after the operation, but it is only fair to recall that for seven months prior to the operation he had had no attacks. After the operation he still complained of the constant dizziness which he described as lightheadedness and which was also present before operation. This steadily diminished after operation, until it was barely noticeable. The patient insisted that this dizziness was always worse in damp weather. It did not seem probable that it was caused by the disturbance of the eighth nerve, for it was unaffected by its section and seemed entirely independent of the attacks.

CASE 5.—*History.*—A well nourished man, aged 33, was referred to the hospital in June, 1927, because of dizziness. Two years before, he had suddenly become dizzy while at work. He continued at work but after an hour it was necessary to stop. A month later, a similar attack of dizziness had occurred while he was at work, and it had lasted all day. Nausea or vomiting were not associated with the attack. Objects always rotated from right to left, i. e., toward the deaf ear. During an attack he could not lie on the affected side; the dizziness was less when he was lying on the opposite side. At first the attacks recurred about every month, but there had been one free interval of seven months. During the past year there had rarely been an interval of longer than a week between attacks. In an effort to obtain relief, the nasal sinuses had been operated on, and the gallbladder had been drained. There were some left-sided headaches from the beginning, but they were always more intense just before an attack. A year and a half before admission, loss of hearing was noticed in the left ear; it had steadily progressed. Buzzing and ringing in the left ear had been present constantly for about the same length of time.

Examination.—The results of the physical and neurologic examinations were negative, except for tests of hearing and vestibular function. The audiometer test showed a 58 per cent loss of hearing in the affected (left) ear and 27 per cent in the right ear. In the vestibular tests, caloric irrigations to the left ear did not give any response; they were normal to the right ear.

Operation.—On June, 1927, under ether anesthesia, the left auditory nerve was sectioned at the internal auditory meatus. The patient had an uneventful recovery.

Ten months after the operation, there were no signs of recurrence of the attacks. There was occasional tinnitus.

CASE 6.—*History.*—A highly nervous, well nourished woman, aged 54, was referred by Dr. P. S. Sisco of Baltimore. For six years she was subject to violent dizzy spells which came on without warning and were accompanied by nausea and vomiting. The duration of the attacks varied from a few minutes to eight hours. During the attack she was afraid to move, for any change of position exaggerated her symptoms. Her first attack had occurred when she awoke from a sound sleep. Objects moved from left to right, i. e., away from the lesion. The

attacks averaged two or three a month. Vomiting was persistent and exhausting. There was also a distressing desire to micturate and defecate and, since she lay motionless to reduce the dizziness as much as possible, she had to remain on the bedpan throughout the attack. After a seizure she fell asleep, and on awakening felt well again. The attacks had greatly increased in frequency, until she had several each day. A gradual loss of hearing in the left ear had occurred until she thought she was totally deaf on that side. The other ear had not been affected.

She said that for about a year she staggered like a drunken person, but without a definite tendency to fall to either side. During the past month, she had felt

Fig. 3.—Scar after operation at which the eighth nerve was sectioned intracranially for Ménière's disease.

dizzy almost constantly. Recently, any sudden motion of the head precipitated an attack. For years she had been unable to lie on the affected (left) side. Tinnitus—a ringing noise—had been present in the left ear since the onset of the condition six years before. She thought her memory was affected. Occasionally before an attack, she noticed that everything seemed bright, but she did not have a definite visual aura. She also had a vague impression of "smelling something" before an attack, but she could not give any better description of the sensation. There was no history of otitis media.

Examination.—The results of the physical examination were negative. The Wassermann reaction of the blood was negative.

The results of the neurologic examination were negative, with the exception of the changes referable to the eighth nerve.

The hearing test showed a 70 per cent loss of hearing in the left ear and 30 per cent in the right.

The left ear did not show any response to the caloric test; the right ear was normal.

Operation.—On Aug. 16, 1927, the left eighth nerve was divided under ether anesthesia.

The patient left the hospital ten days later. Eight months after the operation, she was well and had had no sign of her old attacks. Only an occasional trace of tinnitus remained.

CASE 7.—*History.*—A woman, aged 35, was referred by Dr. T. P. Sprunt of Baltimore in April, 1927, because of attacks of dizziness. Ten years before a thyroidectomy had been performed by Professor Halsted because of marked symptoms of exophthalmic goiter. She had never been in robust health since the operation, but she had continued to work steadily and had not had any serious illness.

She dated the onset of her condition to a sudden attack of dizziness fifteen months before when she was about to leave home. She had a violent sensation of turning to the right. Shortly afterward she was nauseated and vomited. The attack wore off during the day. Since then, similar attacks had occurred rather frequently, until she had them about every two or three weeks. The dizziness never caused her to reel or fall, but there was the sensation that she was rotating to the right, i. e., away from the lesion. The symptoms were accentuated when she lay on the left side. As long as she kept still and kept her eyes closed, she was much better. Between the attacks she was free from any disturbance. Since the beginning of her dizziness, the hearing in the left ear had been impaired. This had been progressive. Tinnitus was present almost constantly throughout her illness, but was worse, perhaps, at the time of the attacks. Her past history did not shed any light on the cause of her illness. She had never had otitis media or mastoiditis. The blood pressure was 128 systolic and 82 diastolic. The Wassermann reaction of the blood was negative.

Examination.—The results of the physical and neurologic examinations were negative, with the exception of the local alteration in the function of the acoustic nerve.

The audiometer test showed a 56 per cent loss of hearing in the left ear; the right was normal. The caloric tests showed a normal response in both ears.

Operation.—The left auditory nerve was divided intracranially under local anesthesia. When the acoustic nerve was being manipulated and divided, the patient complained of the same sensation of rotation and of nausea as during an attack. It promptly disappeared when the nerve was severed. The postoperative recovery was uneventful.

One year after the operation, the patient had not had any sign of her former attacks. For several months, she had had a feeling of dizziness and uncertainty when walking and was hesitant about crossing the street. This has now disappeared almost entirely. The noises in the ear persist as before. They have become less noticeable as she pays less attention to them. It is my impression that the tinnitus is neurogenic.

CASE 8.—*History.*—A well nourished woman, aged 32, was referred from the medical dispensary. Five years before she was suddenly seized with a severe attack of dizziness accompanied by severe nausea and vomiting. She felt as

though she were "falling forward or backward," and it was necessary for her to hold on to something to keep from falling. The next attack occurred three weeks later. Since then, the attacks had recurred with increasing severity and greater frequency, until they averaged about two or three a week. The attacks occurred without apparent cause and were not induced by movements of the head or body; they often occurred when the patient was sitting or recumbent. Objects moved before her but with indefinite direction; they seemed to whirl in a general confusion. Three years later, she first noticed that the hearing in the left ear was impaired; this progressed until she was practically deaf in that ear. There was no tinnitus. Recently, there were some dull frontal headaches, but they were not important. She said that occasionally, during the most severe attacks, she thought that she lost consciousness, momentarily, and that she had a tendency to stagger to the right during the dizzy attacks; during the free intervals, the gait was normal. Later a new feature was added to the attacks, in that there was a "queer feeling" in the head just before the attack began. She took this as a warning signal to lie down promptly. There was no history of otitis media or mastoiditis and no known cause for her distress.

Examination.—The results of the physical and neurologic examination were negative except for the local condition.

The hearing tests showed a marked reduction of hearing for all tones in the left ear. Air conduction was greater than bone conduction. Hearing on the right side was essentially normal; there was about 65 per cent loss of hearing on the left by the audiometer test and 18 per cent on the right.

In the caloric tests, irrigation of the left (affected) ear with ice water caused rotary nystagmus. The response of the right ear was more active.

Dr. Crowe had observed this patient for more than a year, and had made several audiometer examinations. The tests showed a steady increase in deafness.

Operation.—The left auditory nerve was divided intracranially under ether anesthesia. The postoperative course was uneventful. Four months after the operation, there had been no suggestion of an attack. It is worthy of note that one year before the operation, the eighth nerve was explored and an anomalous artery lying on the nerve was "clipped." A unilateral decompression resulted. No improvement followed.

CASE 9.—*History.*—A greatly undernourished woman, aged 47, was referred by Dr. J. Heyward Gibbes of Columbia, S. C., on Jan. 2, 1928. Five years before, she was suddenly seized with an attack of dizziness while walking. She did not fall, but probably would have done so if she had not sat down. She was nauseated and vomited. Since then, similar attacks, lasting from three to five hours, had occurred every few weeks, but not infrequently they had occurred every two or three days. After many spells, she remained very weak for several days; at times it would require two weeks to recuperate. During the attacks, everything seemed to be turning around from right to left, i. e., toward the affected side. She could lie comfortably only on the back of her head. There was one free interval of six months, and for the past nine months nausea and vomiting had been absent during the attacks. She fell during three seizures. She said that the attacks occurred so suddenly that she fell before she realized she was dizzy. The seizures varied considerably in severity. Diminution of hearing was first noticed in the left ear at the time of the first attack. The hearing steadily diminished, until she was practically deaf in that ear, but the hearing was unchanged on the other side. Tinnitus had been present almost continuously in the left ear since the first attack of dizziness but was never present in the right ear. She described the sensation as a buzzing sound; at other times it was like a

whistle. It was always worse after a dizzy attack. She did not think that the hearing was worse after the spells. Headaches did not occur. She had not had otitis media.

Examination.—The results of the physical examination were negative. The Wassermann reaction of the blood was negative.

The neurologic examination showed the following positive symptoms: The left corneal reflex was greatly diminished; the Romberg sign was positive, the patient falling to the left. Left facial weakness (but doubtful) was suggested by the fact that blinking seemed a little tardy. There was no ataxia or adiadokokinesis. The eyegrounds were normal.

The audiometer test showed 90 per cent deafness in the left ear. Bone conduction was absent for tuning forks 128 to 512. Hearing in the right ear was normal. Weber's test referred to the right ear.

The caloric test did not show any subjective or objective response whatever on the left; the right ear was normal.

The absence of response to caloric stimulation, the positive Romberg sign and the diminished corneal reflex made me entertain the diagnosis of a cerebellopontile tumor, though the character of the attacks seemed more like Ménière's disease.

Operation.—Cerebellar exploration was performed on Jan. 4, 1928. There was no tumor in the cerebellopontile angle. The auditory nerve was clearly visible from the pons to the internal auditory meatus. There was no sign of pressure in the posterior cranial fossa. The auditory nerve was divided.

The patient made an uneventful recovery and was discharged two weeks after the operation. There have been no attacks to date (three months after operation). The patient wrote that she was in better health than for many years. Occasional tinnitus occurred.

SYMPTOMS AND DIAGNOSIS OF MÉNIÈRE'S DISEASE

There has not been the semblance of an attack since operation in any of the nine cases. There was reason to question the cure by the operation in only one case (*2*). This patient is the only one of the nine who had total loss of both vestibular and auditory function (at least to our tests) at the time of the operation. It is, therefore, open to question why she had attacks if the nerve was totally out of commission. We are thrown on two possible explanations: (1) that some slight nerve function might still have been present and have evaded the tests, or (*2*) that since the last attack before operation the deafness had become complete, and if this were true, the cure should and might well have been spontaneous and not dependent on surgical division of the nerve.

It may be asked how it can be certain the nine cases are Ménière's disease. The diagnosis was made on the objective symptoms of unilateral subtotal deafness, and in each instance, inspection of the entire intracranial course of the auditory nerve excluded a tumor or other localized space-occupying lesion. Perhaps the most important single diagnostic feature of Ménière's disease is the sudden, fulminating onset of "attacks" without warning and without recognizable inciting cause; and after the attack has subsided, the patient is again in perfect health with

only a residual unilateral loss of hearing and persisting tinnitus. It will be seen from table 1 that while there are differences in detail, the general character of the attacks is much the same. Dizziness and unilateral tinnitus are the only two absolutely constant symptoms in the cases of this small series. In one patient nausea and vomiting were absent in all attacks, and in another, the nausea and vomiting, though present in early attacks, later disappeared. The character of the dizziness varies. Usually but not invariably objects rotate or whirl. Objects may seem to turn in one direction as in case 1, to the left; they may turn first one way then another (case 3), or in no definite direction (cases 7 and 9). In one case (7), the patient seemed to be turning to the right, and in another case (3), she seemed to revolve first one way then another. It is interesting to note that in three cases objects seemed to rotate toward the affected side and in two toward the opposite side.

During an attack, movements of the head intensified the dizziness in all cases. In one case, increase of symptoms resulted from the movement of the eyes so that the patient had to keep her eyes shut during the seizure. Nearly all patients are at once forced to seek the recumbent position. One patient dared not move from the flat of her back or turn her head until the attack had worn off. Two other patients could not lie on either side and obtained relative freedom only when lying on the back of the head. One patient was not conscious of any difference in symptoms owing to position. Without exception, movements of the head or body failed to induce dizziness after the attack had passed, though one patient always slept on the opposite side because she feared an oncoming attack.

Tinnitus was uniformly present and always in the affected ear. In one case (8), however, it did not appear until five years after the onset of the attacks and even then was a minor symptom. Usually it persists in the free interval between the attacks. At times intensification of the tinnitus seems to usher in an attack and is worse during and for sometime after the seizure has passed. Tinnitus is described as a ringing or buzzing sensation. In one case, tinnitus did not develop until six months after the first attack and was synchronous with deafness.

Hearing was diminished in one ear in every case, usually to the point of rendering the ear almost useless for practical purposes. In recent cases in which audiometer tests were made, the loss of hearing was 75, 65 and 90 per cent. It is noteworthy that although the loss of hearing was noticed synchronously with the first attack, this is not necessarily true. In case 2 deafness was not noticed until two years after the first attack; in case 4, five months, and in case 8, three years after the first initial seizure. It is, of course, possible that the early stages of deafness were not observed by the patients. It has been noted already that total deafness was present in only one patient from this series. In the other cases, the losses of hearing were 56, 58, 65, 75 and 90 per cent. In three

TABLE 1.—*Data Showing Gen*

Case	Sex*	Age	Side Involved	Duration of Symptoms	Duration of Hearing Loss	Dizziness	Tinnitus	Nausea	Vomiting	Self or Objects Turning	During Attacks Dizz Made Worse by Lyin	
											Affected Side	On Oppo Side
1	♂	53	Left	2½ yr.	2½ yr.	+	+ Buzzing and ringing	+	+	Objects turn to left	.....	.....
2	♀	54	Left	3 yr.	1 yr.	+	+ Ringing; constant 3 months	+	+	.....	More comfortable on affected side	+
3	♀	55	Right	1½ yr.	1½ yr.	+	+ Continual ringing, worse just before each attack	+	+++	+ Objects turn first one way then another; also feels as if she is turning	Comfortable only when lying on back of head	Cannot on eith side
4	♂	47	Left	1 yr.	6 mo.	+	+ Tinnitus began six months after first attack	+	+	No	No	No
5	♂	33	Left	2 yr.	1½ yr.	+	+ buzzing and ringing	No	No	Objects right to left	Made dizziness worse	More com fortable o opposite side
6	♀	54	Left	6 yr.	6 yr.	+	+	+	+++	+ Objects turn without consistent direction though often left to right	For years had been unable to lie on affected side (left)	.....
7	♀	35	Left	15 mo.	15 mo.	+	+ Worse in attacks	+	+	Sensation that she is turning to the right	Changes make dizziness worse	No differ ence
8	♀	32	Left	5 yr.	2 yr.	+	+ But only in later months; never a conspicuous symptom	+	+	Sensation that she was rocking back and forth; objects also whirl but without definite direction	Position makes no difference, all comfortable	No diffe ence
9	♀	47	Left	5 yr.	5 yr.	+	+ Almost continuous	+ But recently absent	..	Objects turn from right to left	Can lie only on back of head	Can lie o on back head; b tween attack can lie with com fort onl on oppo site side

* In this column, ♂ indicates male; ♀, female.

Do Symptoms Increase by Movement of Head	On Affected Side: Caloric Test	On Affected Side: Hearing Test	Other Signs and Symptoms	History of Otitis Media	Wassermann	Remarks	Time Since Cutting Eighth Nerve	Result
....	Normal	Marked impairment	Headache during attacks	No	Negative	Tinnitus increased in attacks; bradycardia (60) in attacks	3½ yr.	Well; no attacks
....	Total absence of response	Total deafness	Zigzag flashes of light, migraine? pain in left occipital region; tingling and numbness of hand; unsteady gait	No	Negative	.....	3½ yr.	No attacks; well, except for attacks of migraine which have been present for 25 years
+	Normal	Marked impairment	.....	No	Negative	Some ringing and slight loss of hearing in opposite ear	14 mo.	No attacks; well; ringing in ear when she gets tired
+	Normal	Diminution of hearing	Left Babinski sign; left ankle clonus	No	Negative	Patient has constant dizzy sensation between attacks	1 yr.	Well; no attacks; constant tinnitus persists
—	No response	58% loss (also 27% loss in good ear)	Some left sided headaches in attacks	No	Negative	.....	10 mo.	Well; no attacks; occasional tinnitus
++ Must lie on back and perfectly still	Total absence of response	75% loss (also 30% in good ear)	Distressing desire to urinate and defecate during attacks; tendency to stagger; sees lights before attacks	No	Negative	.....	10 mo.	Well; no attacks; no tendency to stagger now; tinnitus at times
Symptoms improved by keeping eyes closed	Normal response	50% loss	Patient had some residual symptoms of exophthalmic goiter	No	Negative	Sensation of whirling and nausea when acoustic nerve was divided (under procaine hydrochloride)	1 yr.	Well; no attacks; tinnitus persists
+	Response is less acute than in other ear	Loss 65% (18% loss in good ear)	Patient thought there was momentary loss of consciousness in attacks; "queer" feeling in hand became premonitory symptom	No	Negative	.....	4 mo.	Well; no attacks; no tinnitus
+	No response	90% loss	Left corneal reflex diminished; Romberg sign, falls to left	No	Negative	Tinnitus was always worse after dizzy spell; attacks come so suddenly does not realize she is dizzy until after she has been thrown to the ground; exhausted for a week or more after attacks	2½ mo.	Well; no attacks; tinnitus persists, but is not so annoying as before operation

cases there were losses of 18, 27 and 30 per cent in the good ears. These losses may or may not have any relationship to the other ear, which is the participant in Ménière's syndrome.

One of the most surprising observations is the great variability in the results of tests of vestibular function. If Ménière's disease is really a primary lesion of the semicircular canals, it is fair to expect the vestibular function to be profoundly and uniformly disturbed; at least, it should be more disturbed than the auditory function. On the contrary, the acuity of response from caloric stimuli is found undiminished in three cases, and only slightly diminished in another.

Three of the patients in the foregoing case reports had headache during the attacks. In two cases, the headache was on the side of the lesion and more in the occipital region than elsewhere. In another instance, the headache was general. One patient had a "queer" feeling in the head just before an attack. She looked on this as an infallible warning sign.

One patient thought there might have been on one occasion momentary loss of consciousness. In none of the remaining cases was there a suggestion of loss of consciousness. The preservation of consciousness is indeed one of the striking characteristics of Ménière's disease. A distressing desire to urinate and defecate during an attack made it necessary for one patient to lie on the bedpan until the attack was over. Two patients complained of some general dizziness or "light headedness" between the seizures. In both of these cases the dizziness persisted for several months after the eighth nerve was divided; it gradually abated and in less than a year it had almost completely disappeared. Nystagmus, which is occasionally reported in cases of Ménière's disease, was not observed in any of the cases from this series.

PSEUDO-MÉNIÈRE'S ATTACKS

The question has often been asked whether unilateral deafness is a necessary sign of Ménière's disease. Frankl-Hochwart suggested the term "pseudo-Ménière's" to apply to cases which seem similar but lack the unilateral deafness. The following history is presented as an instance of so-called pseudo-Ménière's disease because the attacks seem similar, yet without any subjective or objective evidence of impaired hearing, and no unilateral signs.

A man, aged 59, referred by Dr. S. J. Crowe, for the past eight or nine years had been having recurring dizzy spells associated with nausea and vomiting. During the attack everything whirled about him. He could not lie on the left side or on the back of the head. The attacks averaged about two a year. There was some tinnitus in both ears. At the onset of his trouble, attacks appeared almost daily for over a month, gradually disappearing. Other spells lasted only a few days, but they were fearful, and he lived in terror of their reappearance. The whirling

objects continued with his eyes shut, but not so severely. Between attacks he was well in every way. Hearing was not impaired and there were no headaches. The results of the physical and neurologic examinations were negative in every respect. Hearing tests were normal in both ears. Caloric tests gave normal response in both ears.

Although pseudo-Ménière's attacks are usually considered to be of hysterical origin, Politzer suggested that in some cases the organic evidence of disturbed hearing may be late in appearing. The analysis of my cases might seem to give support to this view for in three cases the loss of hearing (subjective) was not observed until long after the onset of the dizzy attacks. In the foregoing case, however, the attacks have been present for eight or nine years—far longer than the hearing-free interval in any of my cases. The patient did not have any unilateral manifestations during the attack except that he could not lie on the left side or back of the head. Such a history would seem to preclude a hysterical attack. It is hardly possible that dizziness of this type could be simulated. Moreover, the patient is a practical, placid and unemotional person.

The following report is from the most severe case of dizziness in my experience and differed from all others.

Edema (Encephalitis [?]) of Cerebellum Producing Extreme and Unremitting Dizziness, Also Severe Nausea and Vomiting.—History.—A well nourished woman, aged 43, was referred by Dr. Harry Slack of Baltimore. For three weeks, she had been bedfast with dizziness which was almost constantly present. It was least when she was on her back; became greatly intensified with every movement of the head to either side. Any attempt to raise the head off the pillow instantly brought on such intense dizziness that the patient feared she would pass into coma. She was almost always nauseated and vomited many times. This illness came on suddenly, and without any antecedent illness or infection. She complained of a feeling of pressure in the suboccipital region.

Examination.—The results of the physical and neurologic examinations were normal. There was no ataxia or nystagmus. There was no disturbance of hearing in either ear. Caloric tests gave a normal response on both sides. Cervical rigidity was not noted. The temperature was never elevated. The leukocyte count was 13,400 and 15,800. Lumbar puncture was performed hesitatingly. The pressure was subnormal, the fluid dropping only slowly; the rate of flow was not increased by jugular compression. Examination of the spinal fluid was negative; there were 3 cells and no globulin. The Wassermann reaction of the blood and spinal fluid was negative. The eyegrounds had been closely watched, but always seemed normal until the last four days, when the veins became definitely fuller; the disk margins were unchanged.

Course.—All forms of medical therapy proved unavailing. The patient's condition was rapidly growing worse, presumably because of the inability to take food.

Operation.—On March 17, 1927, a bilateral cerebellar exploration was finally performed because the subjective localized pressure sensation and the type and severity of dizziness made this localization seem most probable. The dura was exceedingly tight. The cisterna magna—a good index of pressure in the posterior

cranial fossa—was obliterated. The tonsils of the cerebellum had herniated into the spinal canal completely filling the foramen magnum. To liberate them, the dura was split in the midline to the atlas. The lateral ventricle was tapped in an effort to decrease the pressure, but there was no hydrocephalus. The cerebellar lobes were equal in size and looked alike; both appeared much smaller than normal. There was no apparent cause of the great pressure. There was no surface appearance of a tumor, and the short duration of such fulminating symptoms seemed to make the presence of a tumor unlikely. An extended search was not made for a tumor. There was no evidence of an inflammatory process. It was difficult to close the muscles owing to the pressure. A bilateral cerebellar decompression remained and constituted the only therapy resulting from the operation. The dizziness at once disappeared and had not returned one year after operation. It is also noteworthy that after the operation the patient could lie in any position without inducing dizziness.

Comment.—The swelling of the cerebellum was undoubtedly due to edema, but the cause of the edema remains obscure. A few months after the operation the decompression was soft and did not protrude. The swelling of the brain, therefore, must have subsided. If a tumor had been present, the decompression would have become progressively tighter. Perhaps in the absence of any recognizable cause the lesion may be considered a form of encephalitis. The cerebellar edema does not suggest in any way angioneurotic edema described by Oppenheim as a not uncommon cause of Ménière's disease. The absence of increased pressure by spinal puncture was due to the fact that the cerebellar tonsils had filled the foramen magnum and prevented the transmission of cranial pressure. The symptoms in this case differ from Ménière's disease in the constancy of the symptoms and the absence of attacks with associated deafness. They differ from pseudo-Ménière's also in the absence of attacks. The symptoms are associated with organic cerebellar lesions, which will be considered shortly.

MÉNIÈRE'S ATTACKS DUE TO A LOCALIZED LESION ON THE AUDITORY NERVE

That attacks with the characteristic symptoms and signs of Ménière's disease may be due to a localized lesion involving the auditory nerve is shown by the following case report:

History.—A rather feeble woman, aged 66, was seen in consultation with Dr. Leslie Gay of Baltimore. Her first symptoms were gradually progressive deafness and tinnitus of the left ear (the right ear was almost deaf from an old infection during childhood). One year later she was suddenly and without warning seized with a terrific attack of dizziness during which there was nausea and repeated vomiting. The room seemed to jump around, the bed to tilt up and the walls to move in every direction. There was no definite revolving sensation. The attack lasted about thirty minutes. Brief spells of much the same character have since occurred almost daily. Any quick movement will induce dizziness. The deafness varied greatly from time to time. Tinnitus was constant in the left ear. Her eyesight became so poor that she could not read. She never had headache.

Her systolic blood pressure varied greatly during Dr. Gray's observations over a period of several months. Usually it ran from 160 to 190, but has been as low as 110. Her hands and feet were swollen at times, even when in bed; at other times, the swelling was absent.

One month before operation her symptoms became much more severe. Roaring was nearly always present throughout the head, and was often referred to both ears. The hearing became much worse. Her gait was unsteady and uncertain. The left corneal reflex was diminished.

Caloric response on affected side was normal. Irrigations started mild attacks of dizziness and nausea.

The audiometer test showed 63.5 per cent loss of hearing in the right ear (old deafness), and 65 per cent loss in the left (affected side).

Operation.—On Nov. 22, 1927, a section of the left auditory nerve was performed. An aneurysm was found under the eighth nerve. It was traced downward and was continuous with the vertebral artery. Two days after operation, the old noises returned. One week later, it was noted that she could not lie on the left side (nerve cut on this side) for any length of time because nausea and vomiting would start, but dizziness had not been present since operation. The patient's condition became slowly worse and the noises became unbearable. She was unable to stand. Three weeks later, the left vertebral artery was tied in the neck. At the same operation, the right vertebral artery was exposed and pinched between the blades of the forceps; death resulted apparently simultaneously and instantly. Immediate release of the forceps was unavailing.

This case is presented as an illustration of a tumor (aneurysm) pressing on the eighth nerve and giving symptoms which, until later neurologic symptoms appeared, did not seem different from those of true Ménière's disease. It will be noted also that the basilar aneurysm was so situated that the vestibular function was not affected, at least as indicated by caloric tests. The case also illustrates the fact that a primary lesion of the semicircular canals or of the cochlea is not necessary for the induction of attacks of the Ménière's type; and that a lesion of the nerve itself and more specifically of the cochlear branch alone can cause them. I am reminded of the analogy with trigeminal or glossopharyngeal neuralgia, in either of which, in their usual form, a satisfying cause is not demonstrable; but occasionally a cerebellopontile tumor causes identically the same paroxysmal ticlike pains.

In this case, moreover, the dizzy spells which were present daily before the operation appeared to stop just as abruptly and as completely after section of the eighth nerve as in the cases of Ménière's disease. It is also worthy of note that noises referred to the ear did not cease. In fact they became distressing and almost unbearable. Noises, therefore, must at times at least, have an explanation other than by nerve transmission.

Another point is brought out in the analysis of the symptoms of this case i.e., that while at first the onset of the attacks was abrupt without warning and not due to postural change, at a later date attacks of dizziness, nausea and vomiting were brought on by quick movements of the

head. In other words, the real difference, if any, between the dizziness in the attacks of Ménière's and that of tumors in the cerebellum and brain stem, need not be great, for one type of dizziness can seemingly merge into the other and either may be induced by the same lesion.

OTHER TYPES OF DIZZINESS FROM KNOWN LESIONS

In order to compare the dizziness during an attack of Ménière's disease with that associated with other known lesions of the brain, and to determine if possible the significance of differing kinds of dizziness, the analysis in table 2 has been made.

It will be seen that seven of these lesions are located in the posterior cranial fossa; only one was found in the right temporal and occipital lobes. It was formerly my impression that if severe dizziness, nausea and vomiting are induced or greatly accentuated when the position of the head is changed, or if the patient is practically unable to lie with the head in certain positions, the lesion, whatever its character, is probably located in the posterior cranial fossa. The fact that this is not absolutely true is shown by case 8 in which the lesion is exclusively cerebral, but there is no doubt that tumors and other lesions in this general region produce dizziness as a symptom much more profoundly and in much more distressing form than tumors located in the cerebral hemisphere.

I had hoped that such a striking symptom as the inability to lie on one side without starting dizziness or without making it worse and the relative or complete immunity from dizziness by lying on the other side might prove to be a valuable localizing sign, or at least indicate the side of the lesion. But this does not appear to be true. From the patients in whom this character of the dizziness has been analyzed and checked with the strictly unilateral lesions, it will be seen that three could lie with comfort on the side of the lesion but could not lie on the contralateral side; and five (one cerebral) could lie in comfort on the contralateral but not on the ipsolateral side. One must, therefore, conclude that this symptom has no importance in deciding the side of the lesion. I believe, however, that this type of dizziness is a most important indication that an organic lesion—a tumor or inflammatory process—does exist in or on the cerebellum or brain stem and possibly certain parts of the cerebral hemispheres. Dizziness does not seem to be different whether tumors are within or without the brain stem and cerebellum. When dizziness is present, there does not appear to be any special feature to differentiate that in Ménière's disease from the dizziness associated with tumors of the cerebellum and brain stem, or perhaps of the cerebral hemisphere. In any of these conditions objects or even the person may move; and in either case change in position of the head may increase the dizziness. On the whole, the dizziness, nausea and vomiting are much more fulminating and profound in Ménière's disease. Moreover, Ménière's is character-

TABLE 2.—*Analysis Showing Different Kinds of Diagnosis in Ménière's Disease*

Patient	Location of Tumor	Kind of Tumor	Patient Can Lie in Comfort on		Dizziness Made Worse by Lying on the			Does Movement of Head Precipitate Dizziness	Do Objects Turn	Does Patient Seem to Turn	Is Onset of Dizziness Sudden or Gradual	Are Attacks Accompanied by			Caloric Test	Hearing	Remarks
			Side of Lesion	Opposite Side	Side of Lesion	Opposite Side	Back					Tinnitus	Nausea	Vomiting			
1	Aneurysm of basilar artery	Aneurysm	No	+	+	Improved	No	Yes	...	...	Sudden	+ Later became roaring and on other side (to lesser extent)	+	+	Normal	65% loss, but varies from day to day	Cutting auditory nerve stopped dizzy spells, but did not stop tinnitus and old noises in ear; diminution of left corneal reflex; blood pressure varies from 110 to 190; gait finally became unsteady, though unaffected at first
2	Left lobe of cerebellum to the left of the center	Sarcoma	No	+	+	Improved	No	Yes	...	...	..	...	0	0	Normal	Normal	Tended to fall to right in attacks (i. e., to opposite side)
3	Right lobe and vermis	Glioma	No	+	+	Improved	..	Yes	...	...	..	...	...	...	Normal	Loss equal on both sides	Tended to fall and stagger to the right
4	Right cerebellopontile angle and lobe	Glioma	+	No	Improved	+	..	Yes	No	No	Sudden	No	...	...	....	Total deafness in right ear	Dizziness was described as a swimming sensation, objects did not move; raising head off pillow brought on dizziness; total deafness of right ear
5	Right extracerebellar	Sarcoma	+	No	Improved	+	..	Yes	...	...	..	No	No	No	Normal	Normal	Tended to fall to the right
6	Left cerebellar lobe	Small tubercle 1.5 by 1.5 cm.	+	No	Improved	+	+	Yes	...	...	..	No	+	+	....	Normal	Unable to turn off left side for five weeks; staggered toward the right; necropsy showed no other tubercles in brain
7	Right cerebellopontile angle	Gumma	No	+	+	Improved	0	Yes	+	0	Sudden	+ Right ear, never left	+	+	Normal	Both ears affected equally	
8	Right temporal and occipital lobes	Cyst with intracystic angioma	+	No	No	No	No	Yes	+	...	Sudden	No	No	No	Normal	Normal	

ized by "attacks" and in the free interval the patient is almost symptom free; whereas from tumors a more or less chronic state of dizziness exists, or at least the patient remains in a potential dizzy state. In true Ménière's disease there is an associated loss of function of the auditory nerve, and this does not obtain in tumors unless this nerve is directly compressed as was true in two of the foreging cases or in occasional cases of cerebellopontile tumors.

It is in the cases of direct involvement of the auditory nerve by tumors that the differential diagnosis from Ménière's disease becomes more difficult. In the later stages of a tumor's growth there are other signs of pressure and of contiguous nerve involvement making the diagnosis easy, but in the earlier stages these manifestations are absent. In neither of the two cases just mentioned were there signs of pressure or of implication of other cranial nerves. On the other hand, in one of the reported cases of Ménière's there was a positive Romberg sign and a diminished corneal reflex, both presumptive signs of a tumor, but a tumor was not present. In another case there was a positive Babinski sign and ankle clonus on the side of the deafness, but seemingly neither was significant. One of the important features of the operation here described is that a tumor if present will be disclosed and if not present the only alternate diagnosis would seem to be Ménière's disease; and for this section of the eighth nerve is the rational treatment.

At times one is confronted by Ménière's disease affecting the only good ear. A case in point was seen by me several months ago and was recently explored. There was some variation from the usual story of Ménière's disease in that the hearing varied greatly from time to time, often suddenly and without apparent explanation. On the chance that a gross lesion might account for this phase of the symptoms—the basilar aneurysm heretofore referred to gave a similar story and was in fact the only case of either series with such variation in hearing—the nerve was explored but there was no tumor. Since it did not seem justifiable to sacrifice his hearing, the auditory nerve was not divided.

In one of the patients from this series, the vestibular and cochlear branches of the auditory nerve were separate and distinct; such an anatomic variation would lend itself to division of the vestibular nerve without injuring the cochlear nerve and the hearing. Even when the two branches are inseparable, an artificial though necessarily inaccurate division should easily be possible with preservation of at least the major part of the cochlear branch. Such a procedure was contemplated but was not carried out in the patient who was deaf in the other ear. Perhaps it may yet be advisable though there is no precedent on which a cure could be assured. Other experiences have shown how sensitive the auditory nerve is to trauma and even though the injury seems slight,

the hearing never returns. An artificial cleavage between the two branches of the nerve, if attempted, must, therefore, be made with the least possible trauma.

IS MÉNIÈRE'S DISEASE "AURAL" VERTIGO?

Grouping of symptoms into a syndrome without an underlying pathologic basis always carries much uncertainty and a burden of proof. On the one hand, it is possible that the same symptoms may be attributable to different causes and on the other hand, different symptoms may have the same underlying cause. It is difficult to know on the basis of what particular symptoms cases may be included or excluded from Ménière's disease.

Apparently no case of Ménière's disease in its chronic form has been subjected to careful pathologic studies after death. This being true it is permissible to doubt that Ménière's disease is really "aural vertigo." There are certain data which at least make it probable that a lesion of the semicircular canals is not the cause. It cannot be reasoned that the lesion is primary in the semicircular canals because movements of the head stimulate dizziness. The examples, par excellence, that dizziness need not be from this source are given in the cases of dizziness due to verified tumors of the brain and allied lesions in the cases already reported. In these cases changes in posture induce dizziness and here the semicircular canals are known not to be involved. Probably stimulation of the semicircular canals induces the dizziness, but the source of the stimulus is elsewhere. It is known to be at a distance and in the nervous pathways, i. e., the cerebellum, brain stem and auditory nerves. These pathways together with the internal ear form a system which when working properly controls equilibrium, and when disturbed induces dizziness. That the semicircular canals do not harbor the primary lesion of Ménière's disease is also strongly indicated by the fact that in one third of the cases reported in my series there was no appreciable alteration of vestibular function as tested by the caloric reaction. I have not felt justified in using other tests of presumed vestibular function, such as the rotation experiments, because of the patient's great dread of inducing attacks.

On the other hand, it may be reasoned that the cases of so-called pseudo-Ménière's disease are precisely like those of true Ménière's except that unilateral deafness is absent, and that in these cases the primary lesion is probably in the semicircular canals and not in the cochlea. Against this argument is the absence of any change in the caloric reactions in the foregoing case. Cases in which there is loss of vestibular function with cochlear function unimpaired have not yet been demonstrated. However, in the absence of positive objective evidence, the location of the offending lesion cannot be assumed.

On the other hand, since in six of nine cases of true Ménière's disease there is loss of both vestibular and cochlear functions, it seems more reasonable to expect the lesion—in these cases at least—to be primary in the nerve itself rather than in the peripheral end-organs. Given the objective evidence of unilateral deafness without vestibular alteration, there are only two possible sources of the offending lesion, namely, the cochlear nerve or the cochlear end-organ. For practical purposes, the exact location does not matter. Section of the acoustic nerve should produce the same result in either point of origin. It is not improbable that the character and location of the cause may not always be the same.

COMPARISON OF MÉNIÈRE'S DISEASE WITH TIC DOULOUREUX AND EPILEPSY

In the symptomatic expression of Ménière's disease one is strongly reminded of other human ills, such as trigeminal neuralgia, glossopharyngeal neuralgia, and possibly of epilepsy. In all there is the same periodicity of attacks, coming on without warning and without apparent cause—all suggesting lesions of nerves or of nerve tracts or systems. In cases of neuralgia, the symptoms remain confined to the domain of the affected nerve, but in epilepsy whole systems, i.e., those controlling consciousness, motor and sensory function, speech, taste and others, may be and usually are involved by the spread of the stimulus. It has been learned from experiments on animals that any cerebral defect is a potential source of epilepsy; that although the cells of the motor cortex are responsible for the remarkable phenomenon of clonic convulsions, lesions of the connecting fibers even far removed from the motor cortex, may induce attacks of precisely the same general character as when the motor cortex is involved directly. Are not Ménière's attacks in reality like seizures of epilepsy (or of trigeminal or glossopharyngeal neuralgia) differing only in that a different and independent part of the nervous system is affected? In either case any defect in the nerve circuit is always a locus minoris resistentiae removing the inhibition which holds functions under control and permitting their explosion as it were. It may be asked whether such a comparison of Ménière's disease with epilepsy is justified because in the former there is always a progressive objective loss of function (hearing). In cases of epilepsy there may or may not be progressing loss of function; the actual result depending on the character of the underlying cause; but regardless of the kind of lesion the expression of the convulsion is just the same. The same is true of trigeminal or glossopharyngeal neuralgia. The characteristic ticlike pain is precisely the same whether a tumor is the offending cause or whether the cause escapes the present inadequate tests.

It is useless to speculate further concerning the character of the lesion causing Ménière's disease, or its precise location within the limits

just defined. Frankl-Hochwart mentioned leukemia, syphilis, rheumatism and trauma as predisposing causes and reported cases to support this statement. Recently I saw a patient (not included in this report) afflicted with Ménière's disease and with proved syphilis of the nervous system. In none of the nine cases in this report has there been any of the predisposing causes mentioned by Frankl-Hochwart. The uniform absence of a history of otitis media or of mastoid infection, or of a positive Wassermann reaction from the blood is worthy of note.

It would seem that the treatment in cases of true Ménière's disease by section of the eighth nerve has precisely the same rationale as section of the sensory root of the trigeminal or glossopharyngeal nerves in tic douloureux. If the attacks begin either in the eighth nerve or in the cochlea—and they begin in one or the other—and if the lesion is strictly unilateral, impulses can no longer be transmitted after section of the nerve. It is then hardly conceivable that future attacks are possible after section of the eighth nerve. Moreover, since section of the nerve is central to the ganglionic cells in the semicircular canals and cochlea, regrowth of the nerve and recurrence of the attacks should be precluded. If these impressions and deductions are correct, section of the eighth nerve should serve as a therapeutic test of Ménière's disease just as section of the trigeminal sensory root or of the glossopharyngeal nerve are tests for tic douloureux of these nerves.

SUMMARY AND CONCLUSIONS

1. Intracranial section of the affected eighth nerve is suggested as a cure for Ménière's disease. This operation has been performed on nine patients, none of whom has had a subsequent attack. The elapsed time since operation varies from three months to three and one-half years.

2. The operation should be attended by no mortality and with no after-effects, since the patients are practically deaf in the affected ear before operation.

3. Although the series of cases is small, the results suggest that section of the acoustic nerve should stop Ménière's attacks just as absolutely as intracranial section of the glossopharyngeal nerve or of the sensory root of the trigeminus stops the attacks of tic douloureux in these two nerves.

4. The symptoms and signs of Ménière's disease are analyzed. The dizziness of Ménière's and pseudo-Ménière's diseases are compared with that of other known lesions—tumors, inflammations and aneurysms—in the cerebellum and brain stem.

5. There appear to be reasons to doubt that the cause of Ménière's disease is primary in the semicircular canals. A primary lesion of the acoustic nerve seems a more probable primary source of the attacks.

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EXPERIMENTAL STUDIES ON DENERVATED LUNGS*

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There is no vital structure in the body that is so frequently the seat of disease as the lung; still the practical importance of the innervation of this organ is not generally appreciated. The complexity and often the contradictory evidence regarding this innervation have prevented the real facts from being disseminated even in medical schools. Recent evidence, however, emphasizes the important rôle which the nerves that lead to the lung may play in clinical disorders. Binger and his associates [1] have shown that the tachypnea which accompanies acute pulmonary disease in the absence of anoxemia is of nervous origin. The cause of sudden death in cases of pulmonary embolism and following thoracentesis, as well as the cause of bronchial asthma and allied conditions, may also be nervous in origin. It has also been suggested that postoperative massive atelectasis (collapse) of the lungs may, in part, be due to a disturbed innervation. Our interest in this matter arose from the hope that following experimental alterations in the nerves that lead to the lungs we might be able to reproduce the latter condition. Our studies deal with the possibility of complete denervation of the lungs, the methods used to produce partial denervation of one lung and the changes in the physiology of respiration that were produced by such denervation. Before entering into the details of this study, it seems wise to outline briefly the anatomy and the histology of the nerves to the lungs.

MACROSCOPIC ANATOMY OF THE NERVES TO THE LUNGS

In man, the bronchi and the lungs receive their innervation from the plexuses of nerves which surround the bronchi in the region of from the vagal nerves and the cervical sympathetic trunk of the corthe hilum of each lung (fig. 1). These plexuses are made up of fibers responding side; schematically, they may be divided into two main plexuses called the anterior and the posterior pulmonary plexuses. It

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1. Binger, C. A.; Boyd, D., and Moore, R. L.: The Effect of Multiple Emboli of the Capillaries and Arterioles of One Lung, J. Exper. Med. **45**: 643, 1927.

has been shown that there is a rich anastomosis between the fibers of the plexuses on the same side as well as between the fibers from the corresponding group on the opposite side.

The Branches of the Vagi that Lead to the Lungs.—The majority of the fibers from the vagi come from the intrathoracic portion of that nerve, while only a few come from the inferior cardiac and

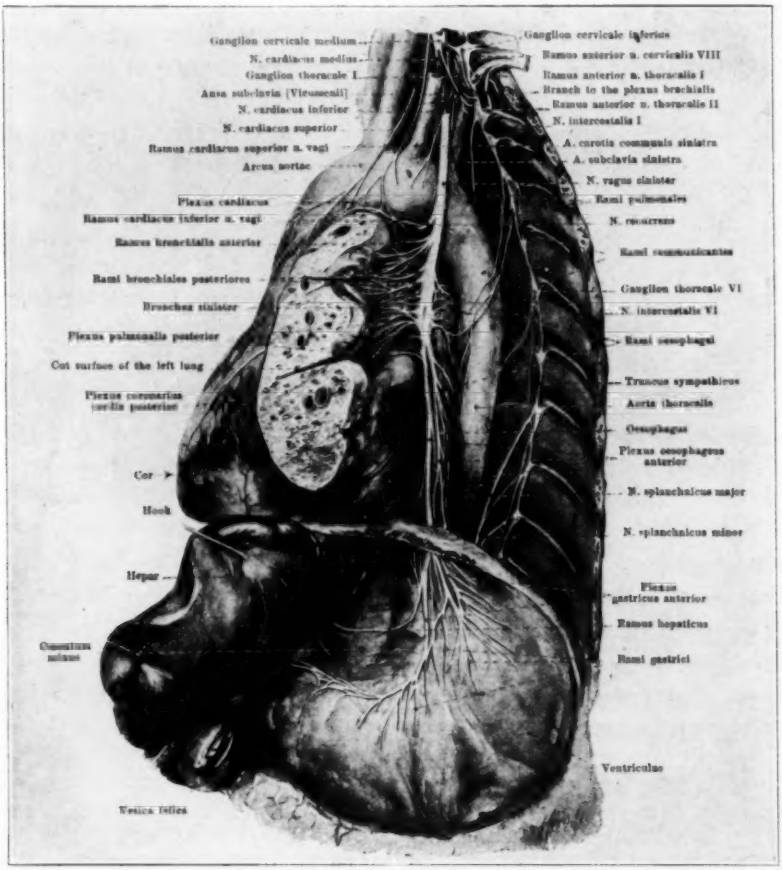

Fig. 1.—The course and branches of the left vagus nerve in the thoracic and abdominal cavities of man (from Spalteholz Atlas, ed. 4).

recurrent laryngeal nerves. The branches from the latter nerves, however, vary greatly in number and in size. From a gross anatomic standpoint, the fibers from the vagal nerves are much more numerous than those from the sympathetic trunks. Hovelacque,[2] in his recent

2. Hovelacque, A.: Anatomie des nerfs crâniens et rachidiens et du système grand sympathique chez l'homme, Paris, Doin, 1927, vol. 1, p. 232.

book, stated that each vagus sends the following branches to the lungs and bronchi:

1. The tracheal branches which form a plexus that surrounds the trachea and both primary bronchi.

2. The anterior pulmonary branches which arise from a small branch of each vagus in its intrathoracic suprabronchial course. The end branches of these nerves enter the hilum of the lungs on the anterior side of the pulmonary vessels. Taft [3] stated that frequently the anterior pulmonary branches arise from the middle, but more constantly from the inferior cardiac nerves.

3. The bronchopulmonary nerves consist of from four to six branches from the main trunk of each vagus. The branches unite to form the anterior and the posterior pulmonary plexuses. Some of these fibers enter the bronchial wall immediately, but the majority follow the posterior wall of the primary bronchi and enter the hilum of the corresponding lung. At this point some of the fibers leave the bronchial wall and course between the visceral pleura and the pulmonary parenchyma for a considerable distance. Other branches from the same plexuses follow the course of the pulmonary veins, and according to Cruveilhier,[4] frequently a small branch is found on the posterior side of the pulmonary arteries.

The Sympathetic Branches that Lead to the Lungs.—There is a rich anastomosis between the sympathetic fibers and the fibers from the vagi so that many of the sympathetic fibers also enter into the formation of the pulmonary plexuses. In general, however, the majority of the sympathetic fibers come from the inferior cervical ganglions and from the first four thoracic sympathetic ganglions. In addition, there are a few fibers from the cardiac nerves which go to the lungs by way of the bronchi and a still smaller group of fibers that accompanies the bronchial arteries.

The Peripheral Ganglions.—In 1840, Remak [5] described the numerous separate ganglions which are interlaced between the fibers that make up the pulmonary plexuses. Later Schiff,[6] Kölliker,[7] Toldt,[8] and Kandarazki [9] showed that these ganglions vary greatly in size and in number, but they are always numerous along the bronchi near the hilum

3. Taft, cited by Hovelacque (footnote 2).

4. Cruveilhier, cited by Hovelacque (footnote 2).

5. Remak, R.; Ztschr. d. Verhandl. f. Heilk., 1840.

6. Schiff: Arch. f. Physiol. Heilk. **6**:792, 1853.

7. Kölliker: Arch. f. mikr. Anat. **2**:320, 1854.

8. Toldt, Carl: Lehrbuch der Gewebelehre mit vorzugsweiser Berücksichtigung des menschlichen Körpers, ed. 3, Stuttgart, 1888.

9. Kandarazki, M.: Ueber die Nerven der Respirationswege, Arch. f. Anat. u. Entwcklngsgesch. **1**:11, 1881.

of each lung. They also showed that some of these ganglions are situated in the superficial layers of the tracheal and bronchial walls, while others are deeply embedded in the submucosa. Izmajloff [10] also described a fine network of nervous fibers in the submucosa of the bronchi which are in direct connection with these ganglions.

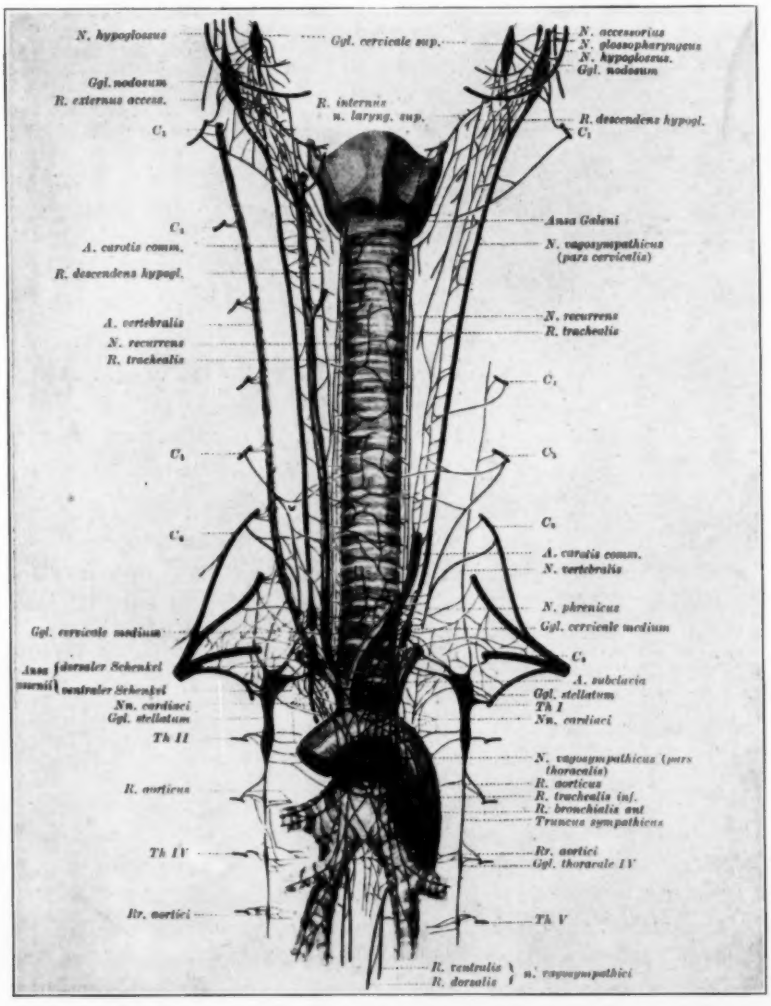

Fig. 2.—Anterior view of the nerves from the tracheobronchial trunks in the dog (from Braeucker [11]).

From the work of the foregoing investigators, it is evident that there is a marked difference between the innervation of the lungs in

10. Izmajloff: Zur Histologie der Nerven in den Atmungsorganen, Thesis, St. Petersburg, Jahresb. f. Anat. u Physiol., 1873, p. 157.

man and in the various experimental animals, yet when we consider all animals in one group we find that there is more variation in the fibers from the sympathetic trunks than there is in the fibers from the vagi. It has been only within recent years that the exact anatomy of the nerves leading to the lungs of the various experimental animals has been worked out. Braeucker [11] added much information to this subject by his work on dogs.

In the dog the nerve supply of the lungs is likewise derived from the vagi and from the cervical sympathetic trunks (figs. 2 and 3). The fibers from the vagi reach the lung by two main routes, namely,

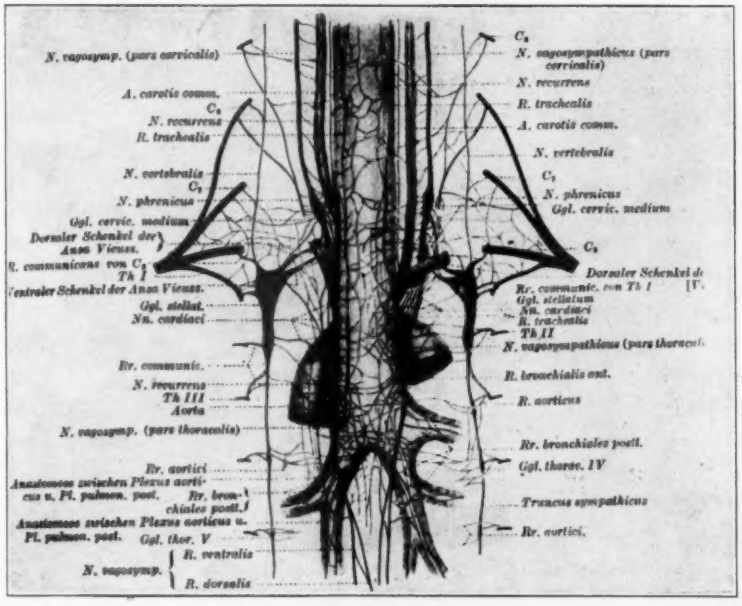

Fig. 3.—Posterior view of the nerves from the tracheobronchial trunks in the dog (from Braeucker [11]).

by the anterior bronchial nerves and by the posterior pulmonary nerves. The former are branches of the inferior tracheal nerves which come from the vagi through the recurrent laryngeal nerves. The posterior pulmonary nerves consist of from four to nine distinct fibers. Three of these fibers are usually well developed and arise from each vagus at the inferior border of the primary bronchi. The branches of the nerves surround the major bronchi and form the posterior pulmonary plexuses. Scattered between the nerve fibers are

11. Braeucker, W.: Die experimentelle Erzungung des Bronchialasthmas und seine operative Beseitigung, Arch. f. klin. chir. **137**:463, 1925; ibid. **139**: 1, 1926.

numerous ganglions of various sizes. The anastomosis with the fibers from the opposite side takes place largely by way of the inferior tracheal nerves. The majority of the sympathetic fibers reach the lung by an indirect route. Some of the sympathetic fibers are distributed to the lungs through the subclavicular plexuses. These plexuses are made up of fibers from the middle cervical [12] and stellate ganglions, from the posterior branch of the ansa Vieusseni and from the rami communicantes of the seventh cervical and the first thoracic ganglions.

Other sympathetic fibers go to the lungs by way of the recurrent laryngeal and the inferior tracheal nerves. Part of these fibers come directly from the middle cervical ganglions, and part of them course through the inferior cardiac nerves. The fibers from the recurrent laryngeal, the inferior tracheal and the inferior cardiac nerves are all grouped together to form the anterior pulmonary plexus, particularly on the right side. The inferior cardiac nerve on that side is made up of two branches from the right vagosympathetic trunk, one branch from the recurrent laryngeal nerve, one from the anterior loop of the ansa Vieusseni, one from the left middle cervical ganglion, one from the left recurrent laryngeal nerve, and one from the left vagosympathetic trunk. All these nerves have a rich anastomosis with both stellate ganglions and the upper thoracic rami communicantes of both sides.

On the left side, the aortic plexus sends numerous fibers to the pulmonary plexuses. The aortic plexus is made up of fine branches from the upper thoracic rami communicantes of both sides (fig. 4).

In the dog, the general distribution of the peripheral ganglions in the walls of the trachea and the bronchi is essentially the same as we have already described for man; consequently, we shall omit the repetition.

In general, there are two groups of nerves which contribute to the innervation of the lungs. The first group consists of fibers from the vagi which course through the bronchopulmonary and the recurrent

12. Braeucker,[11] in his work on the dog, has shown that the ganglion which is included in the common vagosympathetic trunk is in reality the middle cervical ganglion and should always be referred to by that name. Many other investigators, however, refer to that ganglion as the inferior cervical ganglion. We agree with Braeucker that the ganglion which receives the last cervical ramus communicans should be called the inferior cervical ganglion, and the ganglion which receives the first thoracic ramus communicans should be called the first thoracic sympathetic ganglion. In man as well as in the dog, the real inferior cervical ganglion is usually fused with the first thoracic ganglion and it is this association that should be called the stellate ganglion. For this reason the middle ganglion should always be called the middle cervical ganglion, in spite of the fact that it sometimes lies deep in the thorax, even as low as the second rib.

laryngeal nerves. The other group of fibers comes from the middle cervical and the stellate ganglions of the sympathetic trunks by way of the inferior cardiac nerves and by way of the ansa Vieusseni. There are also several small branches which go directly from the thoracic ganglions to the lungs along the course of the bronchial arteries. In the dog, the bronchopulmonary innervation comes from two major plexuses, namely, the anterior pulmonary plexuses and the

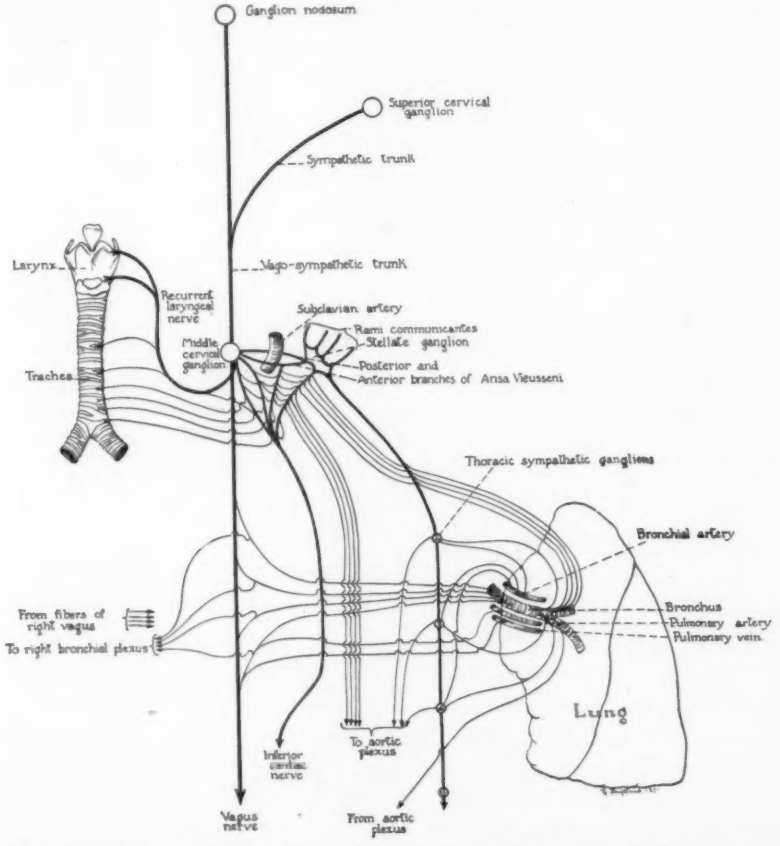

Fig. 4.—Diagram showing the origin of the various sympathetic nerve fibers which go to the left lung of the dog.

posterior pulmonary plexuses. The former are made up of branches from the inferior cardiac nerves together with the inferior tracheal branches of the vagus nerves. The posterior pulmonary plexuses contain only fibers from the vagi. There is a rich anastomosis, however, between the two plexuses as well as between these plexuses and the tracheal and pulmonary plexuses of the opposite side. On the left side, the anterior pulmonary plexus receives fibers directly from the aortic plexus (fig. 4).

MICROSCOPIC ANATOMY OF THE NERVES THAT LEAD TO THE LUNGS

Numerous investigators have carried out extensive studies on the microscopic course of the nerve fibers to the lungs. Braeucker [11] reviewed the work of the various investigators in detail; therefore we shall mention only the conclusions which were reached by their studies. Van Gehuchten and Molhant [13] showed that the bronchomotor fibers of each vagus have their origin in the dorsal nucleus of that nerve, and from that nucleus they extend without interruption to the hilum of each lung. It is still undetermined whether or not these fibers are interrupted in the peripheral ganglions of the lungs, but most investigators agree that it is probable that such an interruption actually takes place. The experimental evidence in favor of this assumption can be found in the report of Möllgaard.[14] He succeeded in completely removing one lung in two young dogs without producing any degeneration of the cells in the dorsal nucleus of either vagus nerve. In these same dogs, he found many degenerated nerve cells in the middle cervical sympathetic ganglion on the side where he had previously performed the pneumectomy. Only a few degenerated cells were found in the middle cervical ganglion of the opposite side. Further studies on the same animals failed to show any direct connection between the nerves to the lungs and the spinal cord. The cells in the stellate ganglions did not show any changes, although a few nerve cells showing marked chromolysis were found in the second and third thoracic sympathetic ganglions.

In similar experiments on cats Möllgaard [14] showed that most of the degenerated nerve cells were found in the stellate ganglions. The middle cervical ganglions are not always present in that animal, but in those animals in which the ganglions were present there was some degeneration of the nerve cells in them following pneumectomy.

From the foregoing experiments, we may draw the following conclusions: There are fibers which go directly from the dorsal nucleus of each vagus to the hilum of the corresponding lung where they are interrupted for the first time in the peripheral ganglions of that lung. The middle cervical and stellate ganglions, together with the second and third thoracic sympathetic ganglions, constitute the sympathetic centers of the lungs (fig. 5).

The Possibility of Denervating One Lung.—After a careful consideration of all the data which we have presented on the macroscopic and microscopic course of the nerves leading to the lungs, the ques-

13. Van Gehuchten, A., and Molhant: Contribution à l'étude anatomique du nerf pheumogastrique chez l'homme, Bull. Acad. roy. de Belgique **25**:859, 1911.

14. Möllgaard, H.: Studien über das respiratorische Nervensystem bei den Wirbeltieren, Skandin. Arch. f. Physiol. **26**:315, 1912

tion arises as to the possibility of completely denervating the lungs of an animal for experimental purposes. Our studies have been made on dogs because the innervation of the lungs in that animal is the most constant, and also because of the accurate data on the anatomy and physiology of the nerves to the lungs that is available.

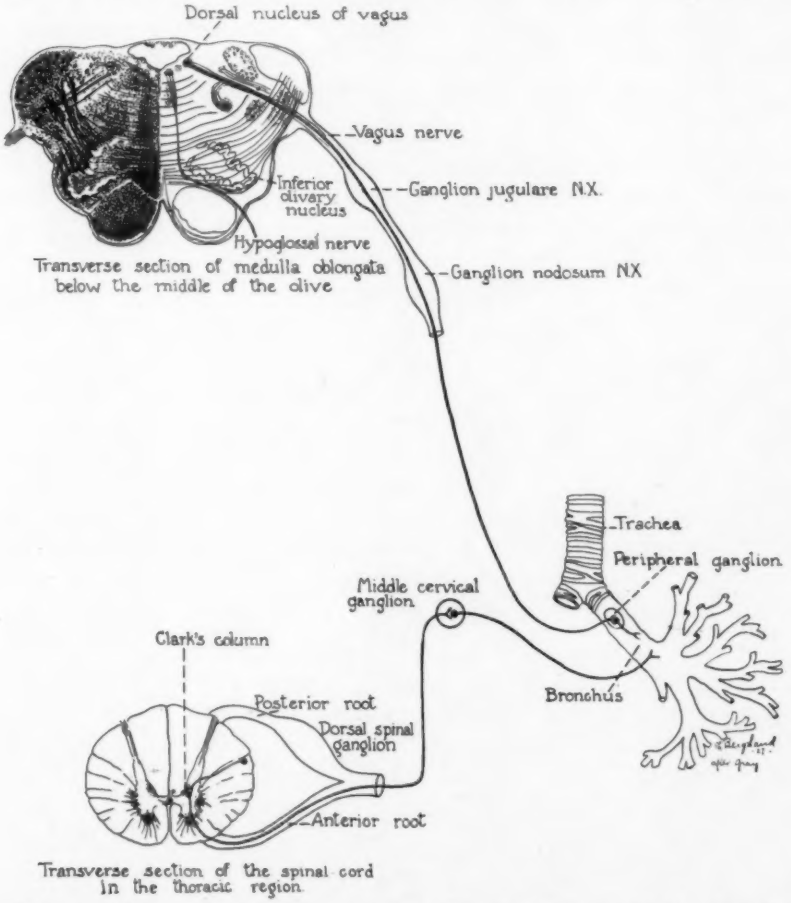

Fig. 5.—Diagram showing the course of the efferent sympathetic nerve fibers to the lung from the nuclei in the medulla oblongata by way of the vagus nerve, and from the nuclei of Clark's column in the spinal cord by way of the thoracic ramus communicans.

From the theoretical standpoint, the lungs can be denervated by two different methods. The first method consists of the removal of all the sympathetic branches to the lung, as well as the removal of a section of the vagus nerve on the same side. The second method consists of the removal of the fibers from the peripheral plexuses at a point just before they enter the hilum of the lung.

The sympathetic fibers to the lungs come from the spinal cord by way of the superior thoracic rami communicantes. The fibers then enter the stellate ganglion and pass through the ansa Vieusseni into the middle cervical ganglions. From there they reach the lungs through the anastomoses which these ganglions have with the recurrent laryngeal and the inferior tracheal nerves, the aortic plexus and the inferior cardiac nerves. Consequently, below the ganglions the pulmonary fibers of the sympathetic trunk are much scattered, and it is difficult to isolate and identify them during any surgical procedure. These fibers are confined to definitely recognizable nerves only in the region of the ganglions, and it is in this region that they should be severed during the operative procedure. Similar results can be accomplished by cutting the rami communicantes of the last cervical and the first seven thoracic sympathetic ganglions. The method of multiple ramisection described by von Gaza [15] would make this procedure an easy one, but, as we have shown, the ramisection would not eliminate the fibers from the various ganglions. Möllgaard [14] showed by his experiments that these ganglions are important centers, and that they are able to function even after they have been completely separated from their central connections. Consequently, we believe that it would be necessary to remove completely the middle cervical ganglion, the stellate ganglion, and the second, third, and fourth thoracic sympathetic ganglions in order to interrupt the direct pathway of the nerves to the lung. In the dog, however, the middle cervical ganglions are usually within the common vagosympathetic trunks; therefore, in order to remove them completely, it is necessary to remove a section from each of the vagosympathetic trunks. It is only in rare cases that the middle cervical ganglions can be separated from the common vagosympathetic trunks. From the experimental standpoint, this fusion of the middle cervical ganglions with their corresponding vagosympathetic trunk is important, since the removal of that ganglion necessitates the removal of a section of the vagus nerve on that side and consequently removes that vagus as a source of nerve supply of the lungs. On each side just above the middle cervical ganglion the recurrent laryngeal nerve becomes separated from the vagus. Thus, by removing a section of the common vagosympathetic trunk for a distance of 1 cm. on either side of the middle cervical ganglion, we simultaneously interrupt the two main nervous pathways to that lung.

From our study of these nerves, we conclude that in order to eliminate simultaneously and as completely as possible the vagus and

15. V. Gaza, W.: Die Resektion der paravertebralen Nerven und die isolierte Durchschneidung des Ramus communicans, Arch. f. klin. Chir. **133**:479, 1924; Klin. Wchnschr. **25**:525, 1924.

the sympathetic part of the innervation of one lung, it is necessary to resect the first four thoracic sympathetic ganglions to remove the ansa Vieusseni and the stellate ganglion, and to resect that portion of the vagosympathetic trunk which has included in it the middle cervical ganglion. All this should be done on the side on which the denervation is to be accomplished.

METHODS FOR SECTIONING THE EXTRINSIC NERVES OF ONE LUNG

The sectioning of the extrinsic nerves of one lung may be done either by the cervical or by the intrathoracic approach.

The Cervical Approach.—In several dogs we succeeded in removing the stellate ganglion by using a cervical approach which was similar to the one outlined by Professor Leriche [16] for the extirpation of that ganglion in man. The approach was made through a low cervical incision just behind the anterior border of the sternocleidomastoid muscle. The stellate ganglion lies immediately beneath the vertebral artery and can be readily exposed by a slight anterior retraction of that artery. From this incision, it is also possible to remove without much difficulty that section of the vagosympathetic trunk which has included in it the middle cervical ganglion. The cervical approach, however, makes the resection of the third and fourth thoracic sympathetic ganglions practically impossible. As we have shown, these thoracic ganglions have numerous important connections with the nerves that lead to the lungs; consequently, those pathways must be interrupted if we hope to produce the denervation of one lung. It is because of this difficulty that we have used the transthoracic route in our studies on dogs.

The Transthoracic Approach.—In man, the thoracic sympathetic ganglions and trunks can readily be reached by an extrapleural approach similar to the operation proposed by Torek [17] for the exposure of the thoracic part of the esophagus. In dogs, however, the extrapleural route would necessitate a long and difficult operation. In our experience, the transthoracic approach is well tolerated by the dogs and it overcomes many of the technical difficulties that are encountered in the other procedures.

TECHNIC OF OPERATION

The dog is first completely anesthetized by the drop ether method, and then a large soft rubber catheter is inserted into the trachea and artificial respiration is instituted by means of the Erlanger insufflation apparatus. The amount of

16. Leriche, R.: De la découverte du ganglion étoilé de des opérations qui se pratiquent à ce niveau, Lyon Chir. **23**:763, 1926.

17. Torek, Franz: The Operative Treatment of Carcinoma of the Oesophagus, Ann. Surg. **61**:385, 1915; Carcinoma of the Thoracic Portion of the Oesophagus, Arch. Surg. **10**:353 (Jan.) 1925.

ether vapor in the system is regulated so that the animal is kept well anesthetized during the entire operation. The dog is then placed on its right side, and the operative field is thoroughly cleansed with alcohol and 1:5,000 mercuric chloride solution. After the sterile linen draping has been applied, an incision about 10 cm. in length is made in the second intercostal space. The incision is carried to the parietal pleura, after which the bleeding vessels are carefully clamped and tied with silk. The parietal pleura is then incised, and the upper lobe of the left lung is retracted to obtain a good exposure in the region of the angle of the second rib. The sympathetic trunk is then visible through the posterior parietal pleura. This pleural covering is incised, and the sympathetic trunk is dissected out to a point well below the fourth thoracic sympathetic ganglion (fig. 6). All the rami of the upper four thoracic ganglions, as well as the rami

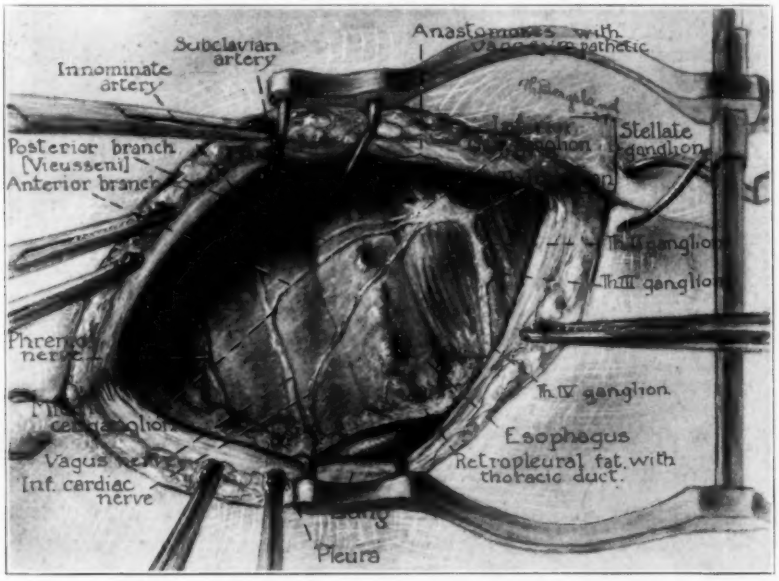

Fig. 6.—Exposure of the stellate ganglion and its branches by the transthoracic route.

of the stellate ganglion are isolated. The excision is begun from below, the sympathetic trunk below the fourth thoracic ganglion being severed first. After all the branches of the fourth, third and second thoracic ganglions have been severed, the lateral branches of the stellate ganglion are isolated and severed. The stellate ganglion is dissected out and the anterior and posterior branches of the ansa Vieusseni are identified and freed. These branches are respectively on the anterior and posterior aspect of the subclavian artery. The posterior branch is cut and the anterior branch is followed to its junction with the middle cervical ganglion in the common vagosympathetic trunk (fig. 7). The portion of the vagosympathetic trunk which has included in it the middle cervical ganglion as well as the origin of the recurrent laryngeal nerve is then completely resected. Hemorrhage during this procedure is practically negligible. Two fine silk sutures are used to approximate the edges of the posterior parietal pleura at the site of the operation. The anterior parietal pleura is closed by

a continuous fine silk suture. The intercostal muscles are closed with interrupted chromic catgut sutures. Just before the second row of interrupted sutures are tied, a hemostat is inserted between the ribs into the pleural cavity. The hemostat is used to spread the soft tissues, and the lungs are inflated to expel the air from the pleural cavity. The subcutaneous tissues are brought together by interrupted silk sutures, and the edges of the skin are approximated in a similar manner.

If peripulmonary sympathectomy is to be performed at the same time as the foregoing procedure, it is better to make the incision in the third intercostal space, since this approach gives a better exposure of the pulmonary artery and

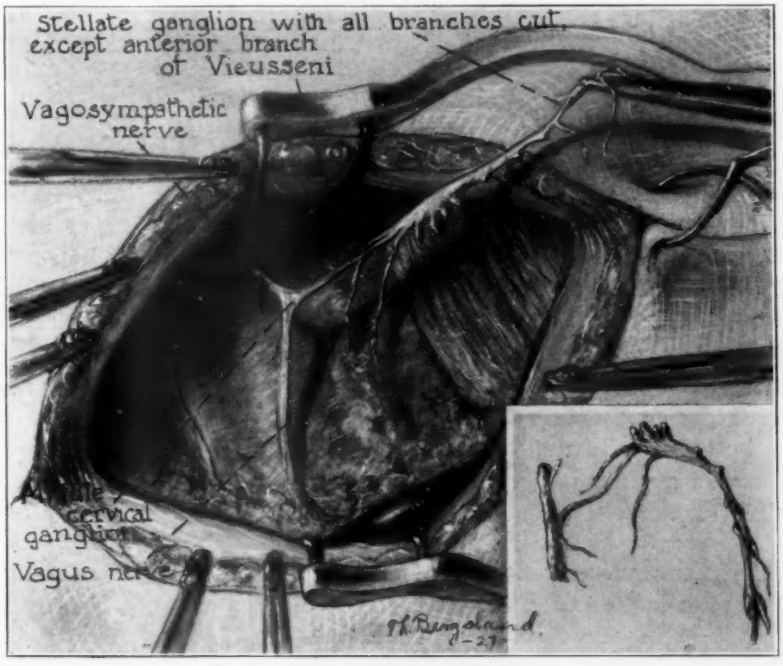

Fig. 7.—Method of resecting the stellate ganglion and its branches. The anterior branch of the ansa Vieusseni is used as a guide to the middle cervical ganglion which is included in the common vagosympathetic trunk.

does not make the resection of the stellate ganglion and its branches any more difficult. We have used the latter procedure on three dogs with satisfactory results.

Comments on the Operative Procedures.—The question arises as to the possibility of producing a complete denervation of one lung. If one recalls the macroscopic and microscopic studies of the nerves that lead to the lungs, one sees that even after such extensive operations as we have already described there are still two sources of nervous impulses that have not been interrupted. The first source is from the ganglions that are situated within the bronchial walls, while the

other source is by way of the fine anastomotic fibers from the pulmonary plexuses of the opposite side. Hence, we must conclude that this surgical procedure does not produce a complete denervation of one lung when the procedure is performed on only one side. In the studies which we were interested in making, it was necessary to keep the nerve supply of one lung intact in order that the reaction of the partially denervated lung could be compared with its normal mate. For this reason, bilateral extirpation of the nerve trunks to the lungs was not used in any of the experiments which we report here. However, even after bilateral extirpation of these nerves there would still be one source of nervous impulses to the lungs, namely, the peripheral ganglions that are situated in the walls of the trachea and bronchi.

Theoretically, it is possible to produce a complete denervation of one lung by removing all the fibers that make up the peripheral pulmonary plexuses on one side. These plexuses, as we have shown, contain all the sympathetic and vagal fibers for one lung. Technically, this is an extremely difficult procedure, since the fibers are so numerous and some of them are so fine that we can never be certain that all the fibers have been interrupted. We have attempted this procedure on several dogs, but after killing the animals and carefully examining the site which had previously been occupied by the pulmonary plexuses, we invariably found several fibers that had not been severed during the operation. We have also tried to denervate one lung by bronchial denudation, but this procedure is likewise an uncertain one in regard to complete interruption of all the nervous pathways to that lung.

RESULTS OF THE STUDIES ON DOGS IN WHICH THE EXTRINSIC NERVES OF ONE LUNG HAD BEEN SEVERED AT OPERATION

A series of ten dogs in which the extrinsic nerves to one lung had been severed was used for these studies. The dogs were observed over periods varying from several weeks to several months and the data which we obtained may be grouped under the following five headings.

Effect on the Respiratory Movements.—The frequency and character of the respiratory movements were charted for several days prior to the operation, and a normal respiratory curve for that animal was plotted. For several weeks following the operation a similar record was kept. In analyzing the various curves, it was found that there was no constant change in the respiratory movements after the extrinsic nerves to one lung had been severed. Likewise, the rate or volume of the pulse following the operation was not altered; the immediate postoperative reaction which usually lasted for a few hours is excluded.

Effect on the Carbon Dioxide Combining Power of the Blood Plasma.—These determinations were made according to the method of Van Slyke and Neill.[18] From the table it is evident that there was not a constant variation in the results following the operation. The slight diminution which followed immediately after the operation usually returned to normal within two days. In some of the dogs, there was a slight increase which persisted over a period of several weeks, but this increase was so slight that it never exceeded the limits of normal variation. The table illustrates the average variation in three different animals as well as the reaction which follows the operation.

Effect of Various Kinds of Experiment Emboli.—In this series of experiments, we compared the reaction of a partially denervated lung with that of its normal mate after various kinds of experimental emboli had been injected into the general circulation by way of the external

Variations in the Carbon Dioxide Combining Power of the Blood Plasma Before and After Partial Denervation of One Lung

Dog W58		Dog W62		Dog W63	
Date	Blood CO_2*	Date	Blood CO_2	Date	Blood CO_2
Preoperative Estimation for Control					
May 28	46.44	June 15	53.50	July 7	47.23
Postoperative Estimations					
May 31	45.46	June 17	53.28	July 11	43.43
June 1	48.12	June 21	54.01	July 11	44.55
June 3	42.82	June 23	51.17	Aug. 6	55.40
June 7	54.41	July 7	53.57	.....	.....
July 7	43.10	.....	.....	.....	.....

* Per 100 c.c., calculated at 0° Centigrade and 760 mm. pressure.

jugular vein. In several of the dogs of this series, we injected 10 cc. of a thick suspension of lycopodium in acacia solution into the left jugular vein. One month after the original injection, the same procedure was repeated, and the dog was killed three days later. Similar experiments were performed with a medium suspension of potato starch cells in saline solution. The general results were the same in both groups of animals. At autopsy, numerous small yellow spots were found distributed equally throughout both lungs. Microscopically, the small yellow areas were shown to be small abscesses. There was no difference between the abscesses in the partially denervated lung in comparison with those in the lung which the nerve supply remained intact (fig. 9).

Effect on the Gross and Microscopic Anatomy of the Lung.—The macroscopic and microscopic studies of the lungs of dogs that were killed at various lengths of time after the section of the extrinsic nerves

18. Van Slyke, D. D., and Neill, J. M., quoted by Macleod, J. J. R.: Physiology and Biochemistry in Modern Medicine, ed. 4, St. Louis, C. V. Mosby, 1922, p. 42; J. Biol. Chem. **61**:543, 1924.

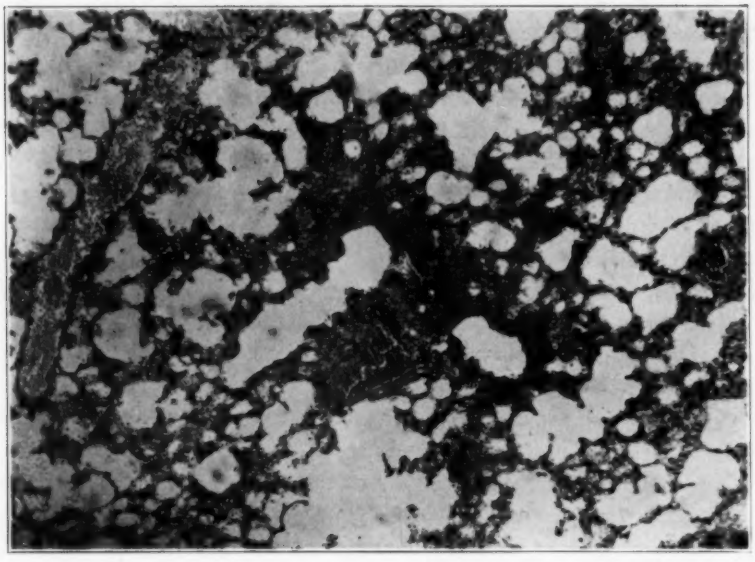

Fig. 8.—Photomicrograph of a section taken from the right lung of a dog in which a suspension of lycopodium and acacia had been injected into the left jugular vein three days previously. Numerous small abscesses were present throughout the entire lung; × 100.

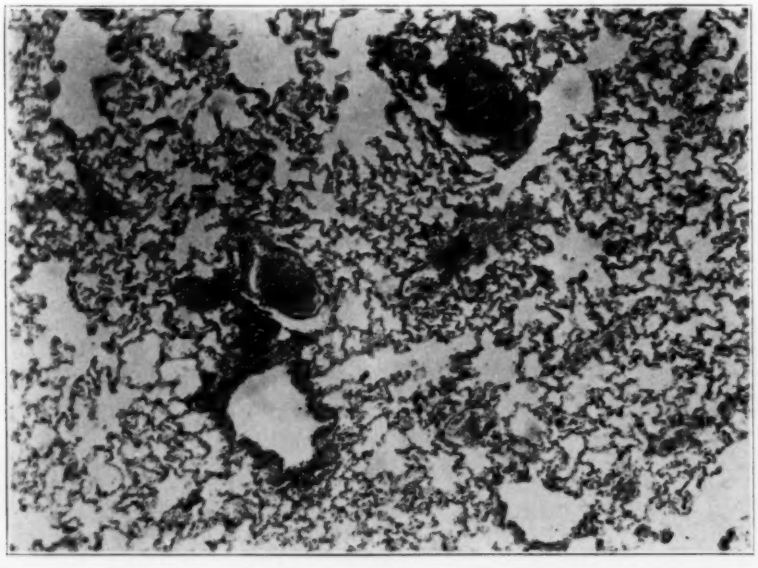

Fig. 9.—Section from the upper lobe of the partially denervated left lung of the same animal as figure 8, showing similar small abscesses; × 100.

to one of the lungs did not show any evidence of changes from the normal. In two of the dogs a few adhesions were found between the lung and the healed operative wound. In none of the dogs were we able to find any evidence of congestion, edema or other pathologic change.

Massive Atelectasis (Collapse) in a Partially Denervated Lung.—One of our dogs developed a typical massive atelectasis (collapse) of the lungs three days after we had severed the extrinsic nerves to the left lung. The immediate postoperative course of this dog was entirely normal. Studies of the carbon dioxide combining power of the blood plasma both before and after the operation did not show any variation from the normal. The frequency and character of the respirations prior to the occurrence of the massive atelectasis were normal. There was no evidence of inflammation about the operative wound. The dog had been active and apparently had not suffered any ill effects from the operation. The animal was placed on the operating table for the purpose of obtaining a specimen of blood for the estimation of the carbon dioxide combining power of the plasma. The dog was quickly turned on its back and immediately became deeply cyanotic; respirations ceased within a short time, and finally the heart ceased beating. A roentgenogram of the chest taken at this time showed the diaphragm on the left side to be higher than it had been on the previous control roentgenograms. Displacement of the mediastinum was not demonstrable in the roentgenograms.

Autopsy revealed a typical massive atelectasis (collapse) of the entire left lung and a partial atelectasis of the lower lobe on the right side. The operative wound was entirely healed. The upper part of the right lung appeared normal. Free fluid was not found in the pleural cavities. In the dog, the mediastinal membrane which separates the two pleural cavities has been shown by Matas [19] and others to be delicate; it is, therefore, fair to assume that when an acute condition such as massive atelectasis occurs on one side, the physical signs which characterize this condition in man may not occur, if the mediastinl membrane ruptures before the heart can be drawn to the affected side. The rupture of this interpleural membrane may explain the difference between the clinical picture of massive atelectasis (collapse) of the lung in man and in the dog.

Macroscopically, all the major and minor bronchi were dissected out, but evidence of obstruction could not be found. The bronchi were normal in appearance and contained practically no secretion. One of the lobes of the collapsed lung was reinsufflated, and the pulmonary

19. Matas, Rudolph: So-called Mediastinal Septum of Dog, in Relation to Pneumothorax Problem in Man, Arch. Surg. **8**:336 (Jan.) 1924.

tissue immediately appeared normal (fig. 10). Microscopic sections from the affected lung showed the histologic picture of complete atelectasis (figs. 11, 12, 13, 14 and 15). Careful search of even the smallest bronchi did not reveal evidence of obstruction or of abnormal accumulation of secretion. The alveolar walls were completely collapsed, and cellular infiltration could not be demonstrated (fig. 15).

COMMENT

In the foregoing case, the massive atelectasis (collapse) of the lung was not due to bronchial obstruction. It is interesting to note that section of the extrinsic nerves to that lung did not prevent the occur-

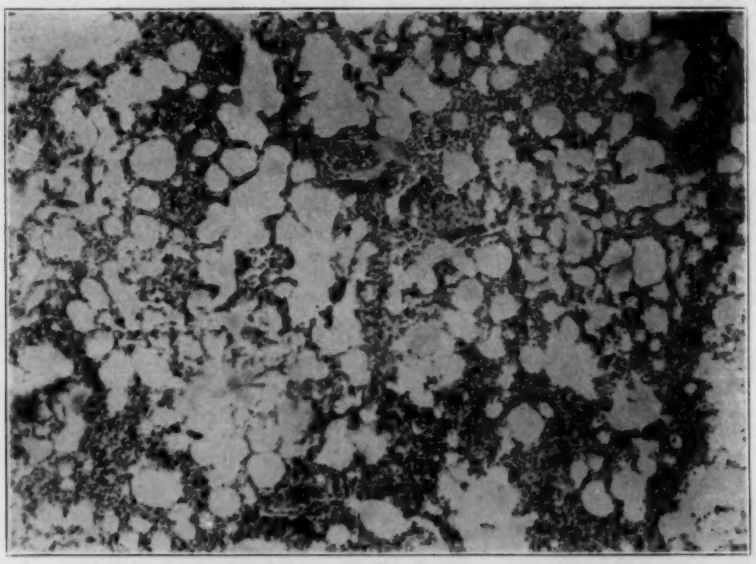

Fig. 10.—Section from the upper lobe of the collapsed left lung after it had been reinsufflated; × 100.

rence of the massive atelectasis (collapse). If there was any reflex responsible for this collapse, the impulses must have come by way of the anastomotic branches from the opposite side, or they must have originated in the peripheral ganglions of the affected lung. It would be interesting to know if after a bilateral section of the extrinsic nerves to the lungs, massive atelectasis (collapse) of either lung could occur. So far we have not been able to produce massive atelectasis (collapse) at will; therefore, further studies along this line cannot be made at this time.

In our series we were unable to show any constant change in the frequency or character of the respiratory movements. Papillian and

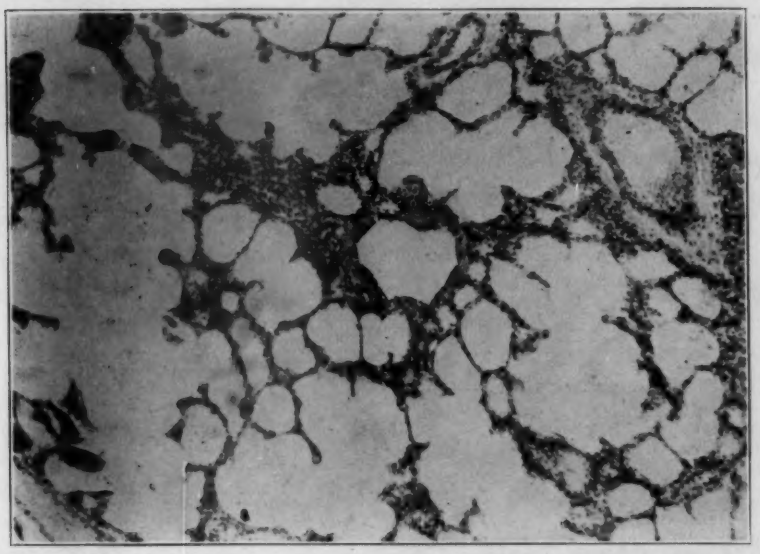

Fig. 11.—Section from the apex of the left lung of another dog, taken during the operation for partial denervation of that lung. Subsequently this dog developed massive atelectasis (collapse) of this lung. Compare with figure 12; × 100.

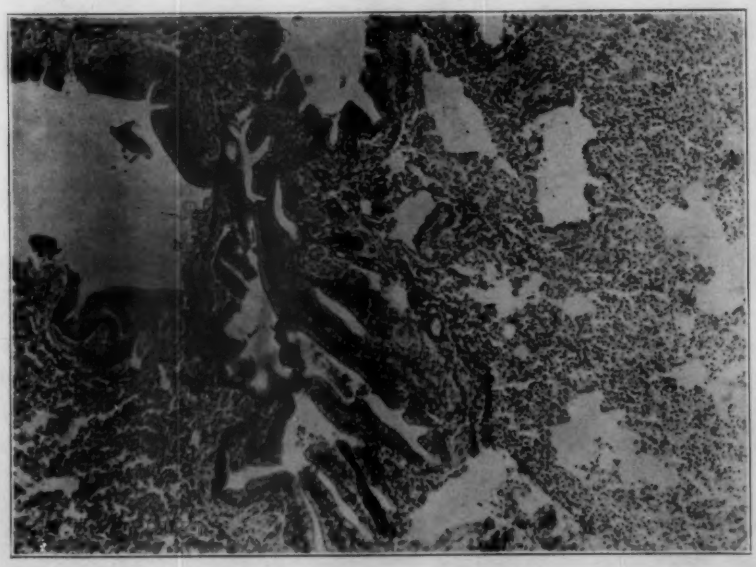

Fig. 12.—Photomicrograph of a section removed at autopsy from the right lower lobe after collapse of the lungs had occurred. Note the collapsed alveoli adjacent to a patent bronchus. This section is from the same animal from which the biopsy shown in figure 11 was taken three days previously; × 100.

Cruceanu,[20] however, stated that after section of the sympathetic trunk on one side, the respiratory rate is diminished for a period of about ten days following the operation. The explanation for this change is still lacking, but, whatever the cause may be, there is no doubt that the animal overcomes the effect. Within a few weeks after the operation, change cannot be detected, and the partially denervated lung reacts as its normal mate to all forms of physiologic and pathologic stimulation.

The heart and the intestine likewise cannot be denervated by section of their extrinsic nerves. Leriche and Fontaine [21] have shown that even the blood vessels have their own intrinsic nervous system. Our studies tend to show that the same condition exists in the lungs, and we are

Fig. 13.—Section from the upper lobe of the left lung of the same animal. There is also a patent bronchus surrounded by completely collapsed alveoli; × 100.

convinced that the intrinsic nervous system of the lungs is able to function independently of the central connections. It is highly probable that all organs of the human body likewise have their intrinsic nervous systems and, under the proper conditions, could be made to function even after they had been separated from their central connections.

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20. Papillian, V., and Cruceanu, H.: Der Einfluss den beiders Cerv. Sympath. auf die Respirationsbewegungen, Clujul Med. **4**:1, 1923.

21. Leriche, R., and Fontaine, R.: Faits chirurgicaux pour servir à la critique des théories actuelles de la vasomotrocité, Presse méd. **31**:481, 1927; Recherches experimentales sur l'innervation vasometrice; le reflexes vasculaires des membres, ibid. **54**:852, 1927.

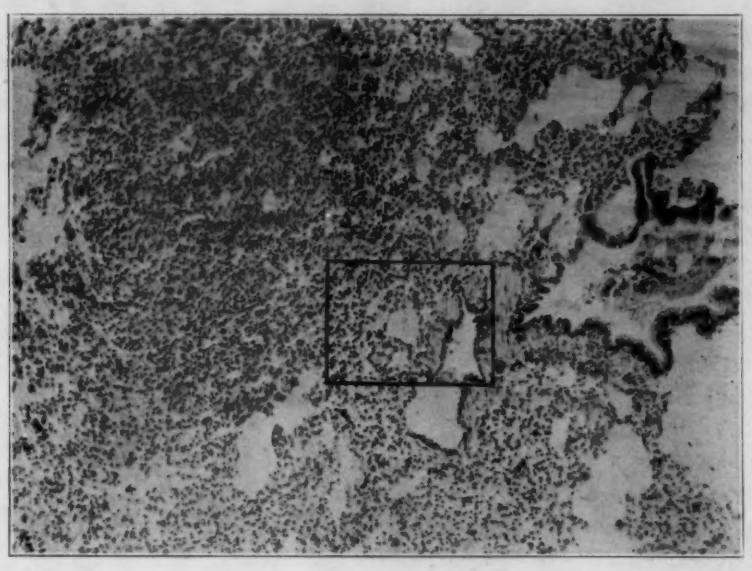

Fig. 14.—Section from the lower lobe of the collapsed left lung; × 100.

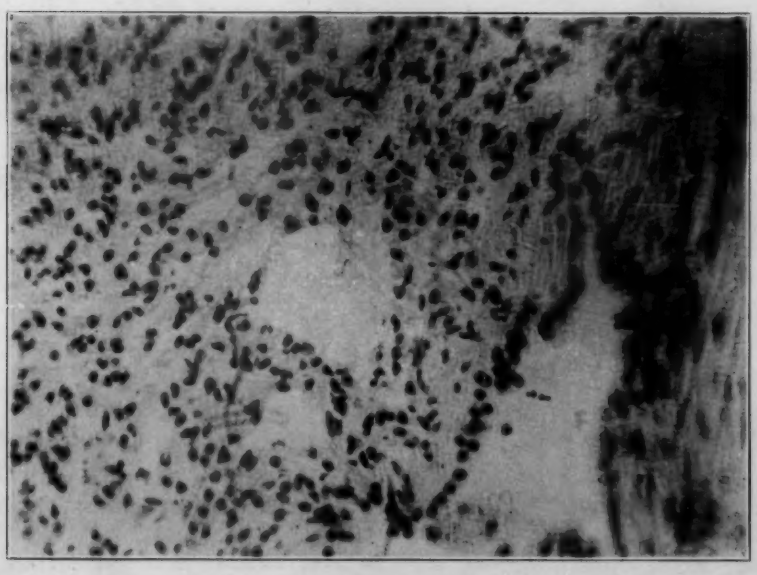

Fig. 15.—High power study of the area included in the square near the center in figure 14. The alveolar walls are completely collapsed, and there is no evidence of cellular infiltration; × 430.

Our experiments tend to throw some light on the explanation for the numerous failures which follow operations on the sympathetic or parasympathetic systems in the surgical treatment of bronchial asthma. We exclude, of course, the failures due to improper diagnosis and improperly performed operations, since these causes for failure have been considered in detail by Leriche and Fontaine.[22] The failures of relief after such operations are still numerous, even in typical cases of asthma in which no special sensitization for foreign matter can be demonstrated. This must be explained on the basis that the reflex paths to the lungs are not completely interrupted. Theoretically, in order to do this, it would be necessary to sever both vagi, to remove both stellate ganglions and to cut the upper four thoracic rami communicantes on both sides. Practically, however, this is impossible in man, since one vagus nerve must always remain intact.

Kümmel [23] first proposed the resection of the cervicothoracic sympathetic trunk for relief from bronchial asthma. Subsequently, he modified this procedure by using the intrathoracic approach and severing part of the branches of the pulmonary plexuses.[24]

Our studies have shown that such a procedure is no better than the simple removal of the stellate ganglion. When we consider that the intrathoracic approach is much more dangerous than the cervical approach, we cannot believe that Kümmel's second procedure has any advantages over his original one. If, in spite of the added risk, one prefers the intrathoracic route, the operation should consist in the removal of the upper part of the thoracic sympathetic trunk, complete extirpation of the stellate ganglion and, finally, section of the vagus nerve on the same side. We believe, however, that a small number of failures will probably always occur after any of the foregoing procedures, in fact even after complete bilateral resection of the extrinsic nerves to the lungs, if that operation could be performed with safety, because of the persistence of the peripheral ganglions situated in the walls of the bronchi. These ganglions probably play an important part in the production of bronchial asthma.

CONCLUSIONS

1. In the dog, partial denervation of one lung is a safe surgical procedure and can be accomplished by unilateral resection of the first four thoracic sympathetic ganglions, the removal of the ansa Vieusseni

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22. Leriche, R., and Fontaine, R.: Sur le traitement chirurgical de l'asthme bronchique, Bull. et mém. Soc. nat. de Chir de Paris **52**:748, 1926.

23. Kümmel, Herman, Sr.: Ursache der Misserfolge bei operativer Behandlung des Bronchialasthmas und ihre Beseitigung, Arch. f. klin. Chir. **142**:499, 1926.

24. Kümmel, Herman, Sr.: Der heutige Stand der chirurgischen Behandlung des Asthma bronchiale, Therap. d. Gegenw. **68**:15, 1927.

and the stellate ganglion together with the resection of that portion of the vagosympathetic trunk which has included in it the middle cervical ganglion.

2. Section of the extrinsic nerves that lead to one lung does not cause any change in the frequency or character of the respiratory movements.

3. The partially denervated lung reacts as its normal mate to all forms of stimulation.

4. The carbon dioxide combining power of the blood plasma remains unchanged after partial denervation of one lung.

5. Section of the extrinsic nerves that lead to one lung does not prevent the occurrence of a typical massive atelectasis (collapse) of that lung.

6. The ganglions situated in the bronchial walls remain intact after the most extensive operation that is directed to produce complete denervation of one lung; consequently, it is technically impossible to obtain a complete denervation of either lung.

7. Because of the persistence of the peripheral ganglions, there will probably always be a few failures from the surgical treatment for bronchial asthma, regardless of which operative procedure has been carried out.

OSTEOCHONDRITIS DISSECANS*

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The present study was made on a specimen removed at autopsy. The patient was reported to have died of diabetes mellitus. During the progress of the autopsy, the knee was discovered to be slightly swollen. Clinical symptoms of trouble with the knee were not recorded, but when the knee was opened, a typical instance of osteochondritis dissecans was found.

The discovery of loose bodies within a joint, especially the knee, has been of great clinical interest, both in respect to the origin of these bodies and as regards treatment. Various names have been used to describe them, such as "joint mouse," "loose body," "floating body" and others. There are several varieties, namely, those which result from a disease process, such as tuberculosis, tabes or pus infection; those which follow definite trauma, wherein a portion of bone and cartilage is broken off; those which result from proliferative changes in the cartilage or synovial membrane as a result of arthritis, and lastly, those in which the origin is obscure or only partially explainable.

It is the latter class which concerns us in this study.

CLINICAL COURSE

In certain instances with or without recognized trauma, patients develop a symptom complex which suggests that there is a loose body in one of the joints. By far the most frequently affected joint is the knee. The symptoms complained of are usually slight swelling, slight discomfort and at times, locking of the joint. Roentgenographic study will at times reveal the loose body, either free in the joint or attached to the articular surface (figs. 1 and 2). Operative removal is indicated. At operation, the loose body is picked out of the articulation: it may be freely movable in the joint cavity, or it may still be resting in its bed. Examination of the body will show a surface of articular cartilage, oval or rounded, with clearcut edge, and an under surface of the subcartilaginous cancellous bone. This body will exactly fit into a defect in the surface of the articular cartilage; the craterlike defect

* From the Harvard Medical School

will also have a clearcut edge of cartilage, with a base more or less filled with fibrous tissue, depending on the period of time which has elapsed since the body fell out of its bed.

This type of loose body in a joint is that which Koenig has described as "osteochondritis dissecans." It occurs most frequently in the internal condyle of the femur, but it also has occurred in the elbow, the hip

Fig. 1.—Osteochondritis dissecans; The loose body is clearly shown in the lateral plane.

and the shoulder joints. It is to be remembered that these joints do not present any other evidence of disease process, nor are there constitutional disturbances present, such as elevation of temperature or illness; locally, signs of inflammatory process, such as heat and redness, are not seen, the pain and swelling present being due to joint irritation of a mechanical nature.

OBSERVATIONS ON THE CAUSATION OF LOOSE BODIES IN JOINTS

About 250 years ago Pechlin studied and, in a way, described the various types of loose bodies in joints. In 1726, Monroe advanced the theory that trauma plays an important rôle in the formation of loose bodies. In 1848, Rainey observed that fragments of cartilage

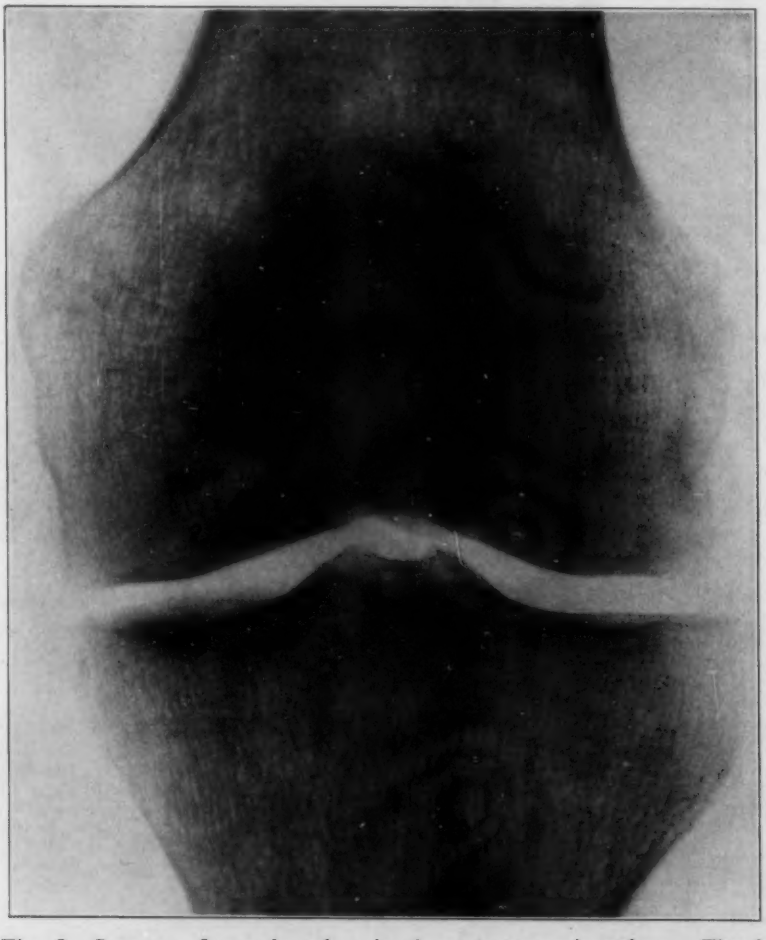

Fig. 2.—Same as figure 1; taken in the anteroposterior plane. The loose body at the surface of the internal condyle is less clearly indicated.

and bone detached within the joint by trauma continued to grow and become sizable loose bodies, regardless of their parent structure. In 1851, Rokitansky stated his belief that bodies composed of bone and cartilage might arise from the articular serosa, representing an excessive development and ossification of isolated nodules of cartilage. John Hunter believed that loose bodies within a joint were at first blood

clots which became organized and attached to articular cartilage, and which assumed its characteristics. Laennec's (1854) theory was that following an arthritis, cartilage might develop in the thickness of the fibrous capsule or in the synovial tissue, and that it might develop the structure of articular cartilage. Paget believed that loose bodies were sequestrums which followed a "quiet necrosis" localized in the base of the articular cartilage; he believed that trauma did not play any part in causation. Journeaux and Hermann (1889) produced evidence of the synovial origin of certain types of loose body, and they believed that synovial cells might take on the characteristics of cartilage cells. Poulet and Villard, at about the same time, expressed the belief that loose bodies in joints were always due to a condition known as "dry arthritis." In 1887, Koenig wrote his description of a distinct pathologic entity which he called "osteochondritis dissecans." He believed that this was an inflammatory lesion of unknown origin, which resulted in the occlusion of the terminal arteries supplying an area of the articular cartilage on the mesial condyle of the femur, producing a separation of a small area of cartilage, and that this area dropped out of the surface of the cartilage with a shell of subchondral bone attached, thus becoming a loose body in the joint space. Koenig realized that trauma might act as a causative factor. His description and explanation aroused considerable controversy. Lexer (1910) advanced the view that certain loose bodies might arise from "embryonic rests" of cartilage cells. Freiburg (1910) reported several well studied cases, and stated his belief that Wollenberg's vascular theory offered a full explanation of the origin of loose bodies. Axhausen (1924) confirmed Koenig's observations as to origin, and drew a forcible comparison between the loose bodies in the knee, in the elbow and in the hip and the osteochondritis known as Legg-Perthe's disease, Köhler's disease of the metatarsal head and tarsal scaphoid. He believes that a slow absorption and necrosis start in the epiphysis and develop into a line of demarcation composed of granulation tissue which is replaced by fibrous tissue. An intercurrent injury jars the loose piece of cartilage loose from its bed. The primary cause is either trauma to smaller blood vessels of the epiphysis or a benign type of emboli lodging in the terminal vessels of the epiphysis.

Timbrell Fisher (1924) showed by experiments on animals that trauma to the epiphysis will produce a low grade type of inflammatory process in a certain area, which thus has lowered vitality and which may be gradually exfoliated. Many observers, notably Codman, have experimented in the production of the actual lesion of osteochondritis dissecans by striking the femoral condyle with a hammer. The lesion may be produced thus in the knee of a cadaver.

Our knowledge of the exact causation, development and character of the lesion known as osteochondritis dissecans is not exact, nor fully explanatory. Since Koenig's original observation, little of importance has been added.

GROSS DESCRIPTION

The articular surfaces of the joint, as a whole, are normal, except for the loose fragment of the internal condyle and slight, irregular linear depressions of the cartilage of the external condyle, which are exaggerated in appearance in the photographs (figs. 3 and 4). There is no lipping of the articular cartilage of tibia, femur and patella, nor any pannus formation.

Fig. 3.—A gross specimen from a case of osteochondritis dissecans; the loose body is resting in its bed.

The loose fragment is attached on its external border to the posterior crucial ligament by a thin band of fibrous tissue. The articular surface of the fragment is roughly a rectangle with rounded corners, 2 cm. in width (internal to the external edge) and 2.5 cm. in length (antero-posterior or sagittal plane). The fragment has a maximum thickness of 8 mm., including the articular cartilage which is from 3 to 4 mm. thick. A prominent feature of the fragment is a transverse depression, shown clearly in figure 2, which divides it into two halves of about equal size. Beneath the cartilage, the fragment when sawn through shows the subchondral bone separated into two parts by loosely textured fibrous tissue. This is shown clearly in the photomicrograph which is

almost a replica of a figure in an article by Axhausen.[1] One cannot escape the conclusion that this buckling was purely a mechanical effect permitted by the character of the tissues deep to the fragment.

On the internal and the external margins, there is a granular cartilage deposit extending for a distance of from 1 to 4 mm. on the deep surface of the fragment, continuous with the articular cartilage of the superficial surface. The reparative growth is not present on the anterior and posterior borders of the fragment.

The depression in the femur is smooth and lined with a thin layer of fibrous material. From both lateral margins there is a thin layer of cartilage extending from the surface downward for a distance of

Fig. 4.—Same as in figure 3.—The loose body is lifted from its bed, and the fibrous attachment to the margins of the internal condyle is shown.

from 3 to 5 mm. On the surface of the condyle at the internal margin of the depression, the articular cartilage shows some fragmentation and separation from the contiguous articular cartilages.

In order to obtain material for microscopic study, a saw cut was made through the internal condyle, passing through the center of the fragment and its socket roughly in the sagittal plane, but at a sufficient angle to avoid continuing the cut into the shaft of the femur. The plane of the section made an angle of 20 degrees with the axis of the shaft of the femur. This cut revealed an irregular cyst filled with soft-textured fibrous tissue lying immediately beneath the crateriform

1. Axhausen: Virchows Arch. f. path. Anat. **252**:515. 1924.

depression. In fact, it was separated from it only by 2 or 3 mm. of cancellous bone. It measured roughly 1.5 by 2.5 cm., and was irregular in outline. At a plane slightly external to this, as revealed in the sectioning of the slab removed for histologic purposes, there was another small cyst which shows well in figure 5.

MICROSCOPIC DESCRIPTION

The crater was lined with a thin layer of poorly-staining, refractive fibrillary material, containing few cells. Its structure indicated that it was probably atypical connective tissue. There was complete absence

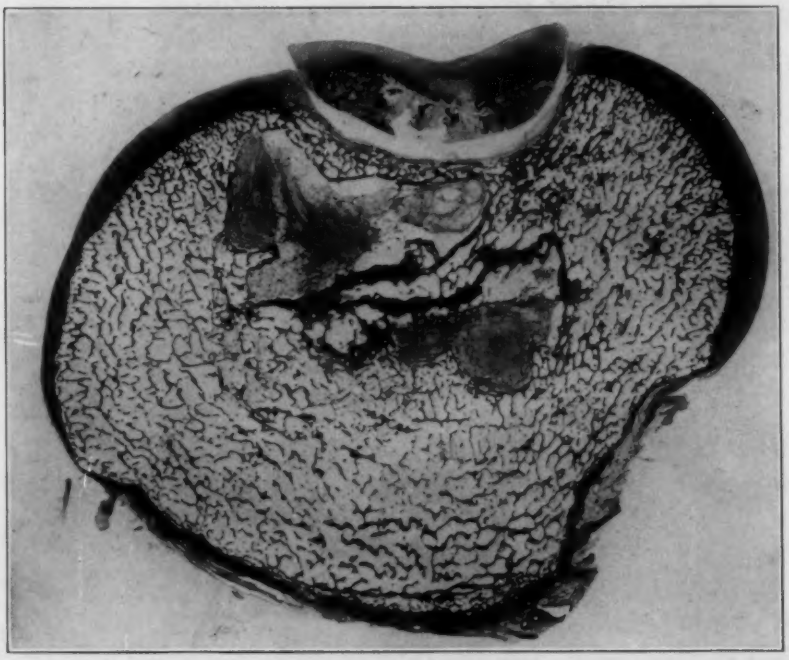

Fig. 5.—Osteochondritis dissecans; low power photomicrograph of a section removed from the specimen which cut through the loose body and the femoral condyle. In this section, the cysts in the condyle lying below the level of the loose body crater are clearly shown. The buckling of the carticular cartilage on the surface of the loose body is evident. Reduced from a magnification of × 1.9 diameters.

of bone repair such as would accompany a fracture or an experimental incision of the bone in animals.

The bone trabeculae of the crater wall, where they were in contact with the lining membrane, were thin and irregular, having the appearance of being excavated on their superior surfaces. There was a zone of deeply basic granular material between the bone and lining membrane, and the impression was that the bone matrix had undergone

autolysis and released its mineral content. Cartilage cells were present here and there throughout in the crater wall close to the bone. Near the periphery or rim of the crater, they formed a thin, definite layer of cartilage with columns of cartilage cells and continuous with the articular cartilage. Thus the impression was forced on us that we were dealing with an unfamiliar reparative process without blood vessel formation, in which cartilage cells apparently originated from bone corpuscles released from the underlying trabeculae.

The cartilage of the fragment appeared to be viable. The cells were normal in appearance. The matrix stained less densely than normally, and this fact, together with the increased thickness of the layer of cartilage as a whole, is probably best interpreted as due to imbition of liquid. The cancellous bone of the fragment was wholly dead. The interstices were filled with basic staining, granular detritus. The bone trabeculae were deeply stained with eosin, and were refractive and without stainable cells. Giant cells or other viable cells were not present.

The "cysts" may be described as regions devoid of bone trabeculae and without specialized boundary zones. The bulk of tissue in these regions was a loose-textured, myxomatous-like connective tissue enclosing small numbers of fat cells. In places, there were dense fibrous tissue bands traversing these regions. At the peripheries there were compact fibrous tissue or merely the normal fat laden cells of the marrow. In many regions, fat cells were separated by faintly staining material containing delicate fibrin strands, migrating polymorphonuclear leukocytes and mononuclear cells—acute changes explained by the presence of blood vessels filled with chains of streptococci, the evidence of a terminal septicemia.

Blood vessels were numerous in these "cysts," except in central regions in which the tissue was compact. Here there were few blood vessels and large extents of tissue had none. Small circular, outlined cavities were found in such regions, the result of necrosis and liquefaction, as shown by the fact that the immediately adjacent fat and loose-textured fibrous tissues were without nuclei and presented other evidences of death. In many places throughout the "cysts" the impression was obtained that the fat cells had undergone necrosis, without decomposition of the fat, and that the myxomatous appearance of the intervening connective tissue was a consequence thereof, and to be interpreted as edema with avascular organization.

Important lesions of arteries and veins were absent throughout the extent of the cancellous bone.

As is obvious from the foregoing account, the histologic observations have not yielded any clue to the processes antecedent to the separation of the fragments. Neither is there any explanation of the origin of the cysts.

A return to the study of gross relationships of fragment and cavity encounters two facts for consideration: the buckling of the fragment also clearly illustrated by Axhausen, and the discrepancy in size of the fragment and the capacity of the cavity. The buckling of the fragment naturally suggests the effects of the yielding of a surface inadequately supported from below. The presence of the "cysts" or areas devoid of bone trabeculae beneath the cartilage naturally suggests the existence of the requisite mechanical factors before the event.

Inspection of the curvature of the articular cartilage in the plane of sections utilized for figure 5 seemed to indicate too great a convexity of the surfaces on each side of the cavity. Therefore, a normal femur was obtained, sectioned in the same place, and superimposed, with the

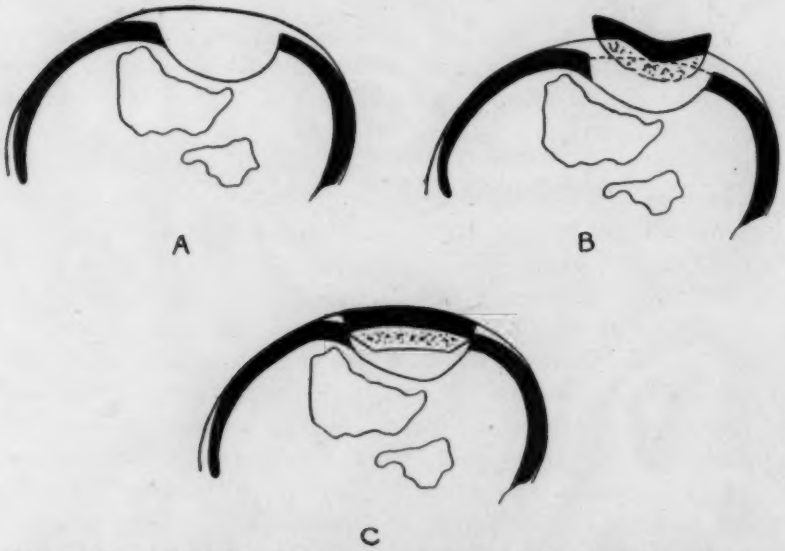

Fig. 6.—Osteochondritis dissecans; comparison of the specimen with the outlines of a normal femur which are superimposed. The probable thickness and curvature of the articular cartilage prior to separation of the loose body is shown in *B*. In *C*, the fragment or loose body is represented as restored to its original contour, revealing an unfilled space which existed before the separation of the fragment.

result shown in the diagram of figure 6*A*. Restorations of the probable thickness and curvature of the articular cartilage prior to the separation is shown in figure 6*B*, the inferior dotted line representing the deep surface of the articular cartilage. In this diagram, the buckled fragment is placed in position with the deep surface of the cartilage coinciding with the reconstructed line of the original deep surface. In figure 6*C*, the fragment is represented as roughly restored to its original contour, revealing a considerable unfilled space and forcing the conclusion that a "cyst" existed in this situation before the separation of

the fragment. The reason for the buckling of the bone and cartilage comprising the fragment is thus accounted for. The change in the curvature of the articular surface of the condyle may also be explained on the basis of the yielding of the bone deprived of support by the absence of a considerable volume of cancellous structure as represented by the several cysts.

The buckling of the fragment probably was the first important mechanical consequence of the change in shape of the cartilage yielding under pressure, and suggests the consequences of lateral stress; a warrantable inference is that the cyst into which the fragment collapsed was originally considerably greater in its lateral (horizontal, anteroposterior) dimensions.

Another inference drawn because of the absence of recognizable new bone formation in the crater is that along the line of separation of the fragment this cyst extended close to the deep surface of the articular cartilage.

On the basis of the microscopic and gross study of the sections, we venture to offer the following explanation of the specimen of obsteochondritis dissecans which came into our hands:

The separation was the effect of mechanical pressure on a portion of the articular cartilage with underlying cancellous bone bridging a "cyst." This pressure probably was intermittent, as occasioned by variation in functional performances of the joint, and probably was the resultant of stresses operating roughly in the long axis of the bone and laterally in the anterior-posterior direction. The lateral stresses are suggested by the change in convexity of the articular surface and the presence of large defects in cancellous bone deep to the lesion. The effects of the loss of the cancellous bone in volume represented by the cysts we think are shown by the shortening of the anteroposterior diameter of the condyle, as shown in figures 6*A*, *B*, and *C*, and by the buckling of the fragment.

There are two essentials to our explanation: one, a sufficient loss in the condyle of cancellous bone to weaken materially the support of the articular cartilage as a whole; the other, the presence of a "cyst" immediately below the articular cartilage in a position subject to vertical and horizontal stresses.

The rarity of typical instances of osteochondritis dissecans of the femur suggests that our explanation, even if dependent on the fortuitous existence and distribution of "cysts," may apply to other examples.

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MIXED TUMORS OF THE THYROID GLAND (FETAL ADENOMAS)

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In presenting this name for a distinct type of tumors of the thyroid gland, I am actuated by a desire to emphasize the clinical importance of these tumors, and to bring them in stronger contrast with the nodulations of old colloid goiters, erroneously called adenomas. If the term "adenoma" can be eliminated from the nomenclature of goiters, it will be easier to attain a true perspective of the pathogenesis of goiters in general. The foregoing designation is, of course, equivalent to the old term "fetal adenoma." The point that it is necessary to emphasize is that they begin as masses of thyroid tissue which has never reached an adult stage. The designation, "mixed tumor," while a strained one, is, in a measure, justified by the life history of the tumor. They have many points in common with the mixed tumor of the parotid and, more particularly, those of the submaxillary glands. Their peculiar topographic relation to the thyroid gland as a whole, their exceedingly slow growth, their relation to the surrounding tissue, the tendency of the connective tissue to increase and, in some areas, to undergo mucoid degeneration, and, finally, after the lapse of decades, to become malignant, alines them in a great measure with the mixed tumors. The formation of cartilage in these tumors is less common than in the mixed tumors of the parotid, but it does occur. They may, like the parotid tumors, develop structures which have been called endotheliomas; this theory is supposed to find its justification in the trabecular or tubular arrangement of the cell columns. As a matter of fact, the cells in the mixed tumors of the parotid gland most likely are epithelial. In this connection it is interesting to note that when these parotid tumors became malignant, they are prone to form acini filled with colloid. It must be admitted, therefore, that the term "mixed" is inappropriate as applied to the tumors of the parotid gland as well as to those of the thyroid gland. The term, however, serves to call attention to a peculiar clinical entity. A satisfactory designation for both must await an extension of the knowledge regarding their genesis. At present, the term more fittingly describes knowledge regarding them than the nature of the tumors. Early tumors of the parotid gland, like those of the thyroid gland, may be composed almost wholly of cells with the containing septums, the "mixed" part being a product of later development. The late development of heavy hyaline bundles

and mucoid tissue in the thyroid tumors does not appear to be a degenerative process, but a part of the life history of the tumor.

The term "mixed tumor" is properly applied to those ovoid growths which are found within the normal or goitrous thyroid gland, the structure of which resembles that of the underdeveloped gland. Its chief gross characteristics are the uniform ovoid shape, the division into renculi-like pyramids, often with central areas of fibrous tissue, and its definite encapsulation.

Closer acquaintance with this tumor is desirable, because it has a life history of its own, and because it has been constantly and persistently confused with the nodules commonly found in lobulated colloid goiters. This confusion has been so complete that many writers believe that the nodulations of the old colloids go through a stage simulating or identical with that of the so-called fetal adenomas. The delusion is at once apparent when the structure of small mixed tumors are compared with the interacinal new formation of glands.

The actual source of these tumors is not known. Clinically, they are commonly seen in young persons, when they are no larger than a hazelnut. Wölfler assumed that they are derived from certain groups of cells of fetal origin situated in the interstices between the acini. Ribbert likewise found cells in this region which he believed gave rise to the tumors. In rare instances, one finds small islands in the interior of goiters of glands typical of these tumors, but whether or not the beginnings of the tumors are in such islands is not known. Most authors who have published illustrations of these alleged islands have presented only high magnifications of a few glands, making it impossible to judge of the topography of the environment. Any one with a willing disposition and a high power lens can prove almost anything particularly if it is "von Verfasser gezeichnet." As a matter of fact, such groups of cells (fig. 1) are frequently found in glands that show a beginning toxicity. But they are multitudinous (fig. 2), and one would have to assume that each of these areas may be the point at which the future adenoma will develop. True enough, areas identical with the fetal adenoma are found in goiters now and then (fig. 3). I have encountered such islands in less than one half of 1 per cent of goiters. Furthermore, most of these little islands showed evidence of degeneration, indicating that they have failed in development and are not potential tumors of magnitude. Those who assume that each bossilation (fig. 7) in their "adenomas" is derived from such an area, hypothecate something unusual in ontogenesis. The multiple origin of true tumors should not be hypothecated without the backing of definite evidence, a thing wholly lacking in this connection.

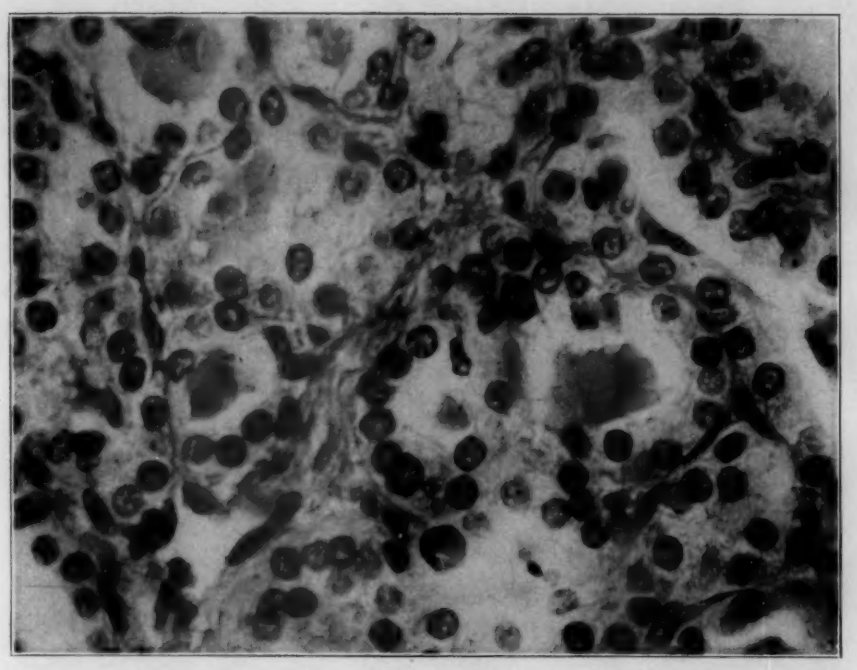

Fig. 1.—Developing acini between acini of a large goiter which was becoming toxic. Note the small acini with little colloid. Compare the nuclei of these cuts with those in figure 8.

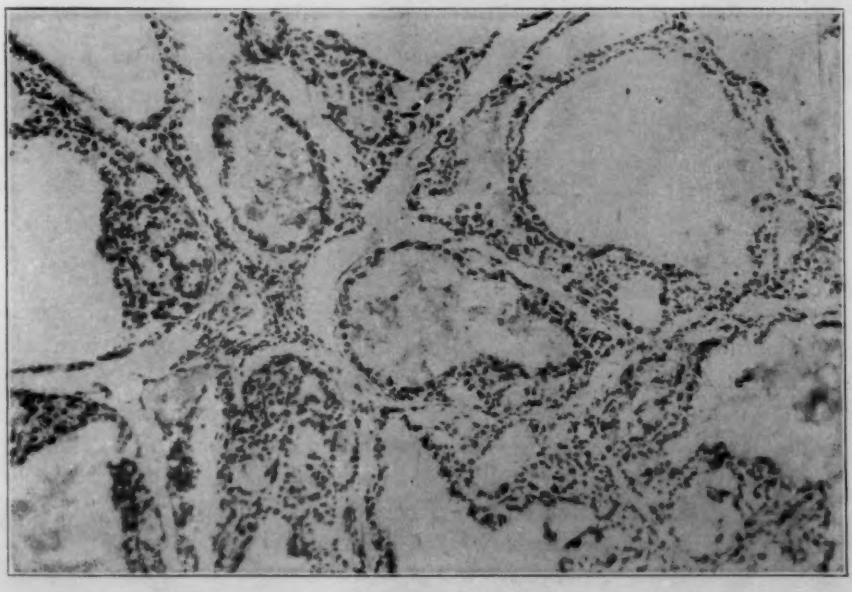

Fig. 2.—Developing acini in the interfollicular areas and in the walls of a pronounced exophthalmic goiter. These areas are obviously hyperplastic.

The development of the common uniform colloid goiter into the nodular type can be easily traced from the formation of new acini in the interstitial cells and from the walls of the old acini, as discussed in my previous paper.

Ribbert declared that fetal adenomas are seldom seen in children. I find them commonly in small children, usually in the absence of enlargement of the gland proper. In the soft goiters of adolescents, they are often conspicuous as small firm nodules easily detected in the softer tissue of the goitrous gland. That they are something apart

Fig. 3.—Small "fetal adenoma" in an old colloid goiter only recently becoming toxic. The nodule, tiny as it is, shows degeneration indicating its age.

from the goiter proper is shown by the fact that they are not influenced by treatment. They never become smaller as the goiter as a whole approaches the normal.

These tumors are nearly always solitary and commonly exist as the only thyroid lesion. Their encapsulation causes them to be definitely separated from the surrounding tissues, causing them to be freely movable. Their capsules, however, are nearly always definitely suffused with the capsule of the thyroid gland. The gland about them is pushed to one side by the growth of the tumor. Because these tumors have been confused with the bossilations of old colloid goiters, this characteristic has been ascribed to all so-called "adenomas." It is only these

Fig. 4.—Cross-section of a typical mixed tumor. The ovoid form, the central fibrous core and the indefinite division into lobules are well shown.

Fig. 5.—The entire tumor and a cross-section of the globular type. The central core is cystic and there is an irregular cystic area to the left of the center.

Fig. 6.—Tumor with extensive central fibrous area with the rencula-like areas about the periphery.

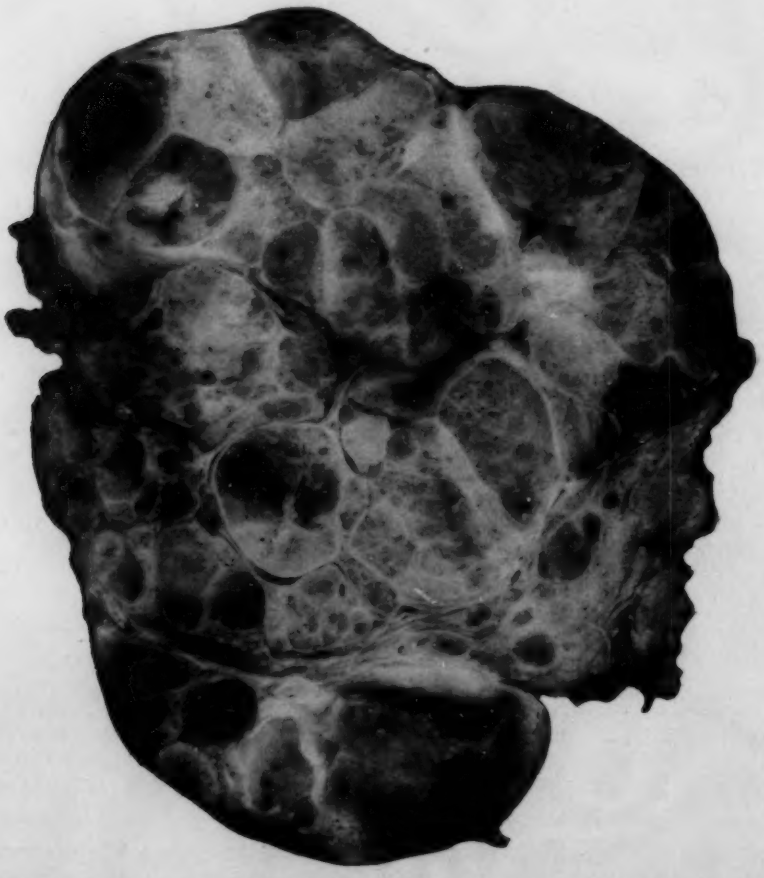

Fig. 7.—Large bossilated colloid goiter showing the innumerable small nodules. In order to fit this into the "adenoma" theory it is necessary to hypothecate a separate focus of origin for each nodule.

mixed tumors that push the gland aside. The bossilations of the old colloid goiters are part of the once diffusely enlarged gland, and there is no normal gland to be pushed aside, or to be saved at operation, as every discerning surgeon knows.

More important than any theory as to origin is the structure of the tumor itself.

PATHOLOGIC ANATOMY

The most striking characteristic of the tumors is their constant ovoid shape, whether they exist alone or as a part of a nodular colloid goiter (fig. 4); less often, they are spherical (fig. 5). They are never lobulated, and they are nearly always solitary. They are firm to the feel and are generally elastic. When they have undergone degeneration, they may be soft or fluctuant.

On section, they are found to be deep red, unless they have undergone secondary degeneration. They are commonly divided into separate pyramidal lobules, the apexes of which point to a common center like the divisions of a grapefruit (fig. 4). In most of them, there is a white core, usually stellate. This core may form only a small nucleus in the center, or it may occupy the larger part of the tumor (fig. 6). This arrangement of the small lobulations with the white center is sufficient to differentiate them from the nodules of a colloid goiter (fig. 7). In many of the older ones various forms of degeneration are noted; this degeneration is usually cystic, but is often colloid and sometimes hemorrhagic. These tumors are always surrounded by a heavy connective tissue capsule, heavier than the true capsule of the normal thyroid gland.

HISTOLOGY

Figure 8 shows the tumor to be made up of closely packed acini. In the younger specimens, few or none contain colloid. The cells are small and closely packed; the nucleus is spheroidal, and the protoplasm, deeply staining. It is interesting to compare the structure of the salivary mixed tumors with such pictures (fig. 9). In the older areas, the colloid is small in amount, acidophilic and without vacuolization, even in specimens fixed in a solution of formaldehyde. As the tumors grow older, the acini may all contain colloid, and the slide may resemble closely the acini of the old colloid goiter (fig. 10).

The most striking and constant picture of these tumors is found in the central stellate area already mentioned. These areas are always made up of heavy bundles of fibrous tissue, resembling keloid, and contain few if any cells. They are faintly acidophilic, and in old specimens, take the dye poorly or not at all. These areas suffuse gradually into the connective tissue between the surrounding acini (fig. 11).

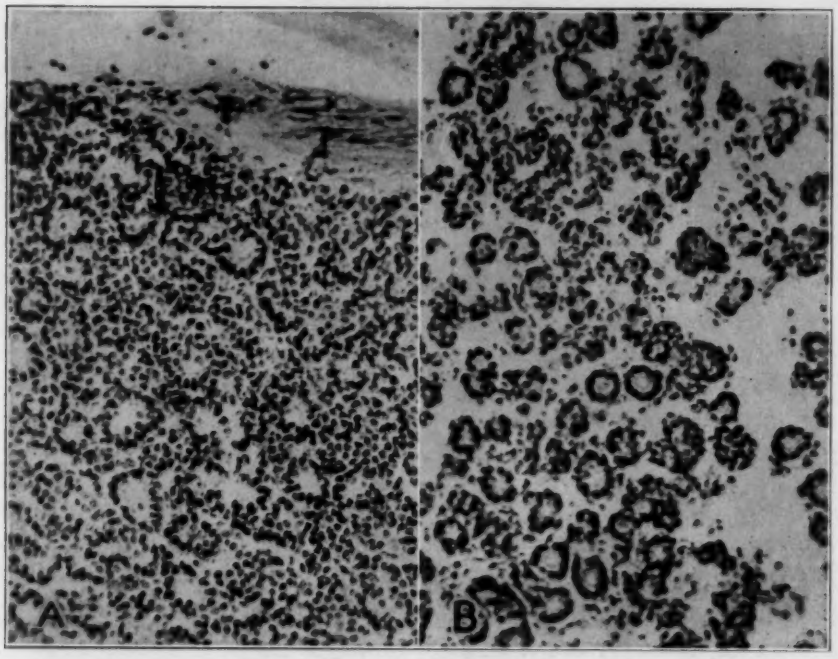

Fig. 8.—Slide from a mixed tumor of ten years' duration. *A* indicates an area near the periphery showing the small acini some of which are beginning to accumulate colloid; *B*, an area nearer the center of the tumor showing the developing mucoid tissue between the acini.

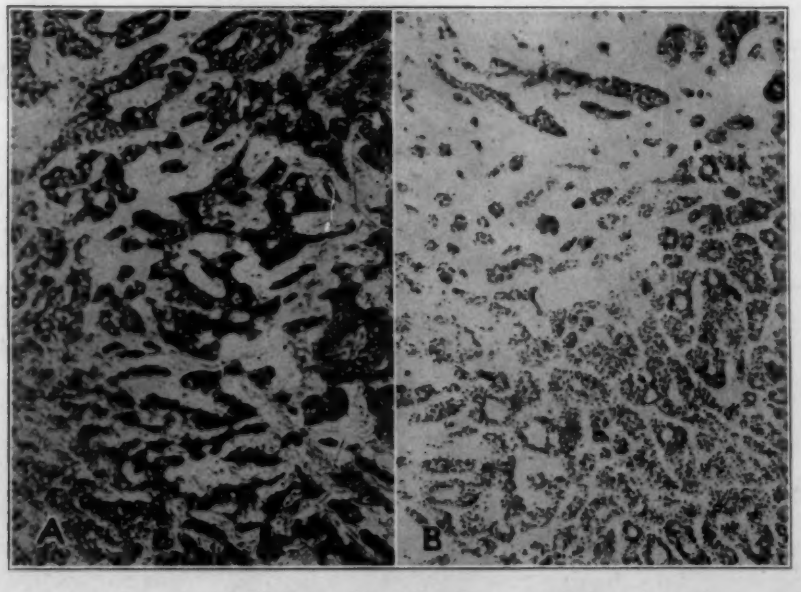

Fig. 9.—Comparison with figure 8; a mixed tumor of the submaxillary gland of eight years' duration. *A* indicates an area near the periphery; *B*, an area near the center showing wide areas of mucoid material.

Wölfler assumes that they result from the organization of blood clots. There is no evidence for this assumption. In the first place, hemorrhages due to a pathologic process do not organize in a regular manner, because they do not form a true clot. Then, too, when blood clots do organize, they do not form bundles such as are seen here continuous with the normal struma of the gland. Furthermore, the fibers of these masses are like the stroma of the tumors in tinctorial reaction. It appears fair to assume that for some reason the tissue forming the core of these tumors is prone to this keloidlike proliferation and forms an essential part of the life history of the tumor.

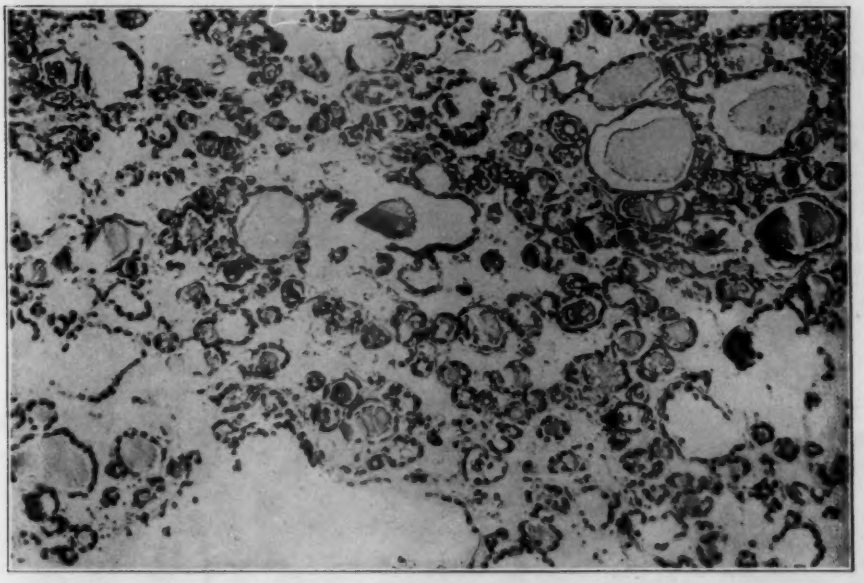

Fig. 10.—Old mixed tumor showing large acini containing colloid. This patient had mild toxic symptoms.

The connective tissue between the acini undergoes a degeneration peculiar to these tumors. It becomes abundant and stains poorly or not at all. The acini are sparse, isolated and often atrophic (fig. 12).

These tumors become larger in the course of time, either by the formation of new acini with accumulated colloid or by the formation of connective tissue or both. In their terminal stages, they may develop degenerative changes and become cystic through softening of the tissue, with or without added hemorrhage. These tumors tend either to a belated imitation of a colloid goiter by developing follicles and the manifestation of hyperactivity or they tend to the formation of malignant tumors.

The gland may take on a belated development resembling that of a colloid goiter. The acini become large and filled with stainable colloid,

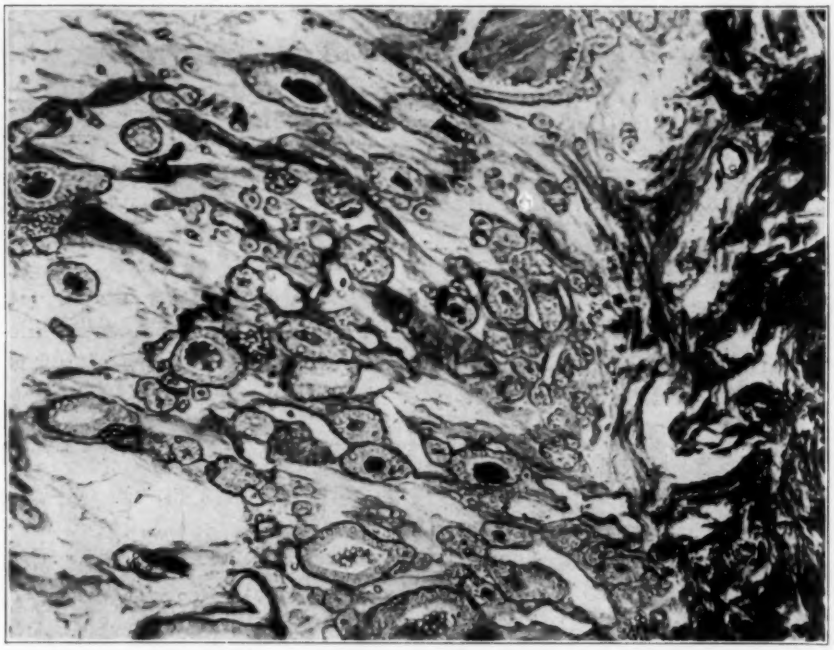

Fig. 11.—Area of the fibrous tissue core showing the heavy fibrin bundles. These fibers are seen to be continuous with the fibers extending between acini with mucoid areas between the fiber bundles (Mallory's phosphotungstic acid stain).

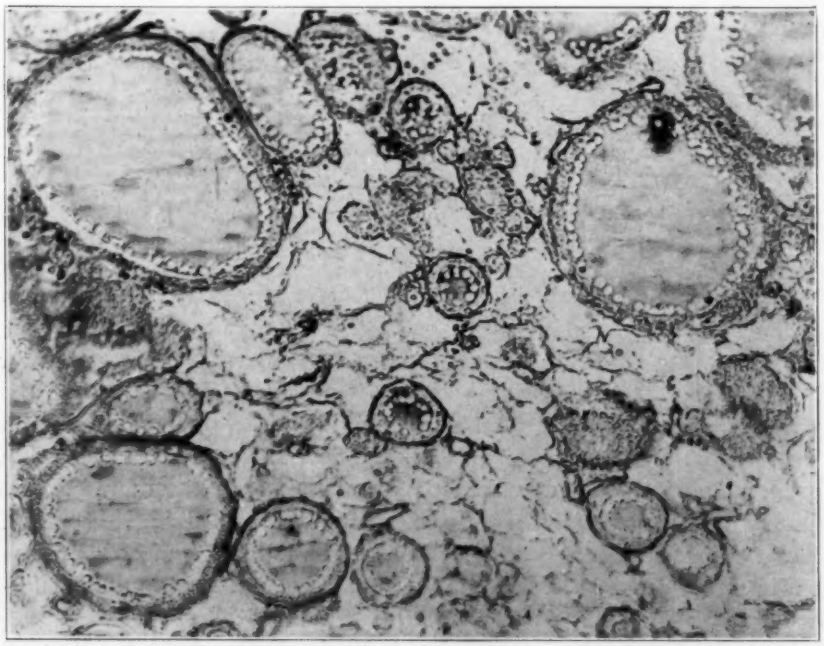

Fig. 12.—Slide showing some large acini filled with colloid together with many smaller ones. Between the acini are fine fibrils the interstices of which is occupied by mucoid material (Mallory's phosphotungstic acid stain).

Fig. 13.—Slide from the periphery of a tumor of many years' duration. The acini have undergone marked proliferation; the capsule was not perforated and the patient has remained free from recurrence, but the tumor must be regarded as histologically malignant.

Fig. 14.—Long straight columns of cells. This tumor had escaped the capsule and death followed by extension.

and new acini may form (fig. 10); the acinal epithelium may even become columnar in rare cases. The colloid never loses its staining characters, nor is there ever any papillation. In other words, these never take on the characteristics of exophthalmic goiter secondarily. Together with these acinal changes, the connective tissue always shows the foregoing peculiar changes, so that there still remains some justification for calling these tumors mixed tumors.

In the second type, the cell columns develop and come to resemble an adenocarcinoma in form. This may occur as rapidly-forming acini (fig. 13) or by forming long columns (fig. 14). This is the type

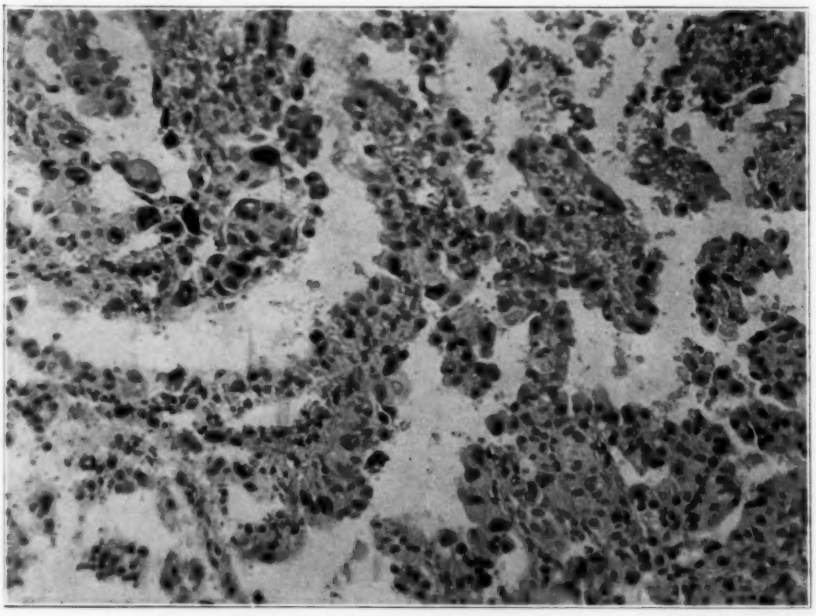

Fig. 15.—The acinal arrangement has been lost. A complete lobectomy removing all obvious tumor tissue failed to cure the patient.

that the laboratory worker is prone to diagnosticate as malignant. The type of connective tissue degeneration which forms a part of the life history of these tumors is strikingly like that seen in artificial tumors (produced by sudan III, tar, etc) and in the initial stages in roentgen-ray epitheliomas. It seems the part of wisdom to regard these tumors as at least potentially malignant when they show epithelial activity, and to govern the therapeutic activities accordingly. The mere fact that permanent cure follows their removal, in most instances before the capsule is invaded, does not invalidate this point of view. The fact remains that when distant metastases occur, this type of lesion is nearly always the cause. Most of the local malignancies are caused

by these tumors. Like the mixed tumors of the salivary glands, these tumors tend ultimately to become malignant. It is only when this fact is recognized that one learns to appreciate the inherent tendency of these tumors.

COMMENT

It is important to emphasize that the tumors are not influenced by medication. This is easy enough to understand when it is remembered that the acini contain little if any colloid. While medication may influence the character of the cells after a prolonged period by causing change in the colloid, it cannot influence the connective tissue framework. This fact seems to have been overlooked by recent students of the influence of compound solution of iodine on toxic glands. Therefore, when one of these nodules is encountered, it should be removed or ignored. The patient may be assured that the removal of the tumor will be necessary some time. The objection to the removal of the tumors in young persons is that a true goiter may develop in later years which will be looked on by the patient as a recurrence. In such cases, it is well to acquaint the patient with this possibility. If treatment is being given for an early colloid goiter which harbors one of the mixed tumors, it is well to tell the patient that while the goiter as a whole will disappear, the nodule will remain unchanged, though it may be less conspicuous by virtue of greater space being provided by the regression of the goiter.

Patients with mixed tumors may be assured that when these tumors become toxic, the toxicity is not extreme, and that eye signs are never produced. These facts may be told patients in the first half of life.

When middle age is approached, while the foregoing facts may be admitted, it must be emphasized that the danger of late heart degeneration, and particularly the development of malignancy, is ever present, and that removal of the tumor is to be urgently recommended.

Furthermore, when these tumors attain considerable size, associated with secondary degeneration, hemorrhage into them occurs; this may result in pronounced inflammation of the capsule and the surrounding tissue and painful affliction. Such a hemorrhage is far more serious when the tumor is associated with a colloid goiter or when it is located in the thorax. In such cases, the sudden augmentation in the size of the tumor may quickly suffocate the patient.

For these reasons, the mixed tumors of the thyroid gland should be looked on as true neoplasms and not as goiters. While these tumors are benign in the earlier stages and allow a certain degree of compromise in treatment, they will cause trouble some time if they are not removed. In this, they aline themselves with many of the milder forms of mixed tumors, notably those of the salivary and mammary glands.

CONCLUSIONS

1. Mixed tumors are true tumors of the thyroid gland.

2. They are comprised of acini more or less characteristic of immature thyroid tissue.

3. The fibrous tissue is prone to have a characteristic keloid-like proliferation and mucoid degeneration.

4. The acini may develop colloid and later undergo the changes of an old colloid goiter, leading to toxicity. The toxicity never reaches the degree of a true exophthalmic goiter.

5. The tumors may menace the life of the patient by a hemorrhage into their substance.

6. They may undergo active acinal proliferation leading to malignancy.

INTRABRONCHIAL POLYPOID ADENOMA

REPORT OF A CASE*

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The development of modern diagnostic methods has greatly stimulated the interest of the clinician in the subject of bronchial neoplasms. That bronchial tumors are by no means an infrequent occurrence has been shown since the roentgen rays and bronchoscopy have come into general use. This is particularly true of malignant tumors. Well localized benign intrabronchial tumors, however, are extremely rare. Since this type of tumor tends to produce obstruction of the lumen which may be followed by extensive pathologic anatomic changes and cause serious symptoms, these cases frequently require the attention of both the internist and the surgeon. For these reasons, the report of a recently observed case seems justified.

REPORT OF A CASE

History.—B. W., a woman, aged 47, was admitted to Mount Sinai Hospital on June 28, 1927. The family history was irrevelant; the past history was negative, except for frequent attacks of tonsillitis in childhood. Five years previous to admission the patient had had pleurisy. Three years previously empyema of the right side of the chest had been diagnosed and subsequently two operations for drainage had been performed. The wound refused to close and drainage persisted until the time of admission. The patient had a persistent cough and expectorated moderate amount of blood-tinged sputum. She said that she has had a bronchial fistula.

At the time of admission, evidence could be found of two operations performed for drainage. There was a fistula discharging considerable pus in the right posterior axillary line at about the level of the eighth rib. Evidence of a bronchial fistula could not be found at that time. Clubbing of fingers was present. On June 30 a roentgen-ray examination, after the injection of iodoform and oil through the fistula, disclosed a small sac below the fistula, but there was no evidence that the iodoform entered the bronchial tree. It was felt, therefore, that there was a chronic abscess of the lung no longer connected with the bronchus, and it was decided to perform a collapse operation to diminish the size of the cavity. The operation was carried out by Dr. Howard Lilienthal on July 1, 1927. Two ribs were resected, and an indurated mass within the lung was felt to which the sinus led. The sinus was split open so that the finger could be inserted, and on removing the finger a profuse hemorrhage of dark red blood appeared under considerable tension which necessitated the termination of the operative procedure. The wound was packed and compression used. Further bleeding did not occur. On July 19, 1927, the deep packing was removed and a distinct bronchial fistula was found. The patient's condition continued to be

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good and she was allowed to walk about. On August 2, while walking, she began to feel weak. The dressings became saturated with blood, and she died almost immediately.

Autopsy Report.—The autopsy was performed the following day by Dr. Paul Klemperer. The most important facts are given from the complete report: There was a healing thoracotomy wound in the right side of the back with resection of

Fig. 1.—The polypoid growth attached to the lateral wall and partly filling the lumen of the bronchus of the lower lobe. The probe is introduced into the tear of the aneurysmatic pulmonary artery branch within the wall of one of the bronchiectatic cavities distal to the neoplasm.

several ribs. In the base of the wound there appeared several lumina from which blood exuded on pressure. The right lung was firmly adherent to the wall of the chest and was removed with difficulty. The right lung weighed 550 Gm., the left, 310 Gm. On opening the lung (from behind), the bronchi of the upper and middle

lobes of the right lung were found to be normal. Within the lumen of the main bronchus of the lower lobe of the right lung there appeared a cylindrical pedunculated tumor which measured 21 mm. by 6 mm. (fig. 1). The tumor was attached to the lateral wall of the bronchus and appeared freely movable. Distal from the tumor there was marked dilatation of the bronchus which opened directly on the surface of the lung and into the wound already described. In

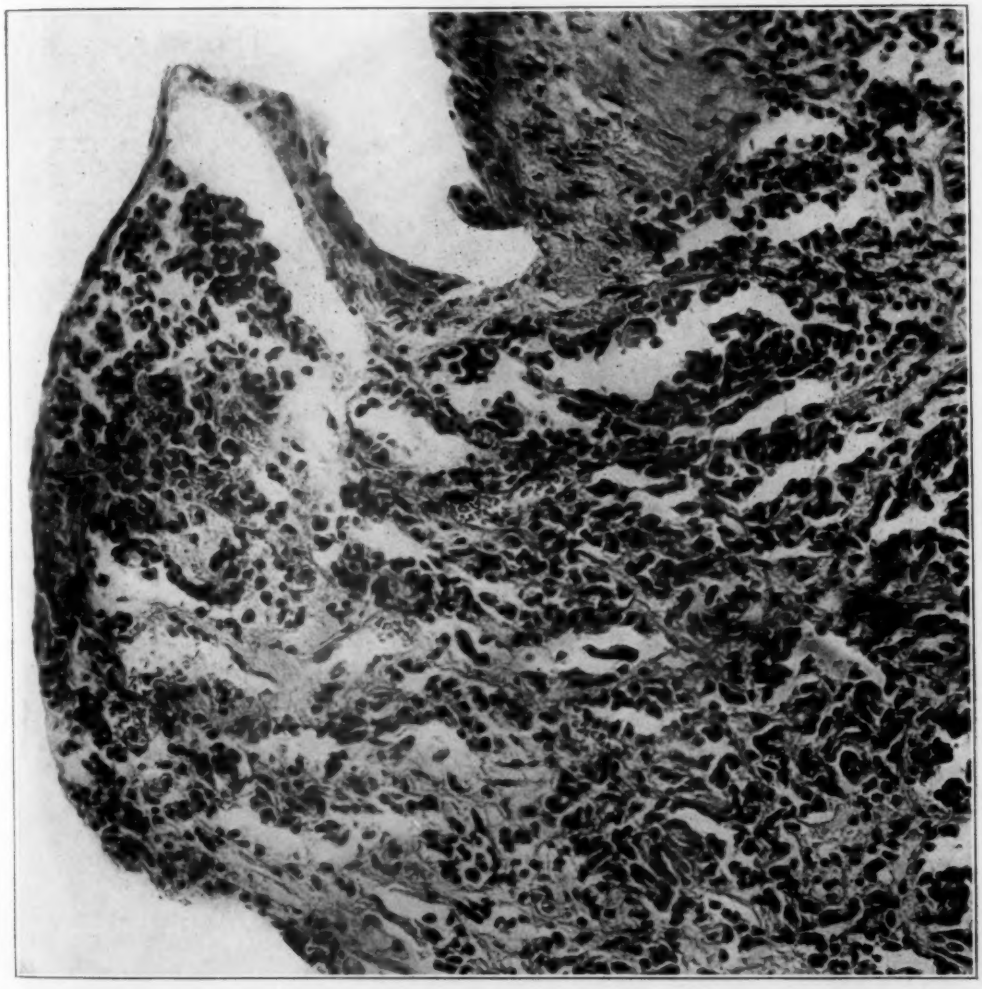

Fig. 2.—Surface of the tumor showing area of stratified squamous epithelium with underlying mucosal stroma. In the other parts, the tumor reaches the superficial basal membrane which is denuded of epithelium; ×80.

following the various ramifications of this bronchus numerous sacculated bronchial dilatations were found, some of which were filled with blood. Within the wall of one of these bronchiectatic cavities there was a blood vessel which showed an irregular tear. A probe introduced into this tear appeared within a branch of the second order of the pulmonary artery. The ruptured portion of the

vessel was an aneurysmal dilatation. In the upper lobe of the right lung and in the left lung no changes were found except aspiration of blood.

Pathologic Anatomic Diagnosis.—The diagnosis was as follows: polypoid tumor of the main bronchus of the lower lobe of the right lung with partial occlusion of the lumen; multiple sacculated bronchiectases in the distal ramifications of the bronchus with bronchopleural fistulae, and rupture of an aneurysmal dilatation of a pulmonary artery branch with subsequent severe hemorrhage.

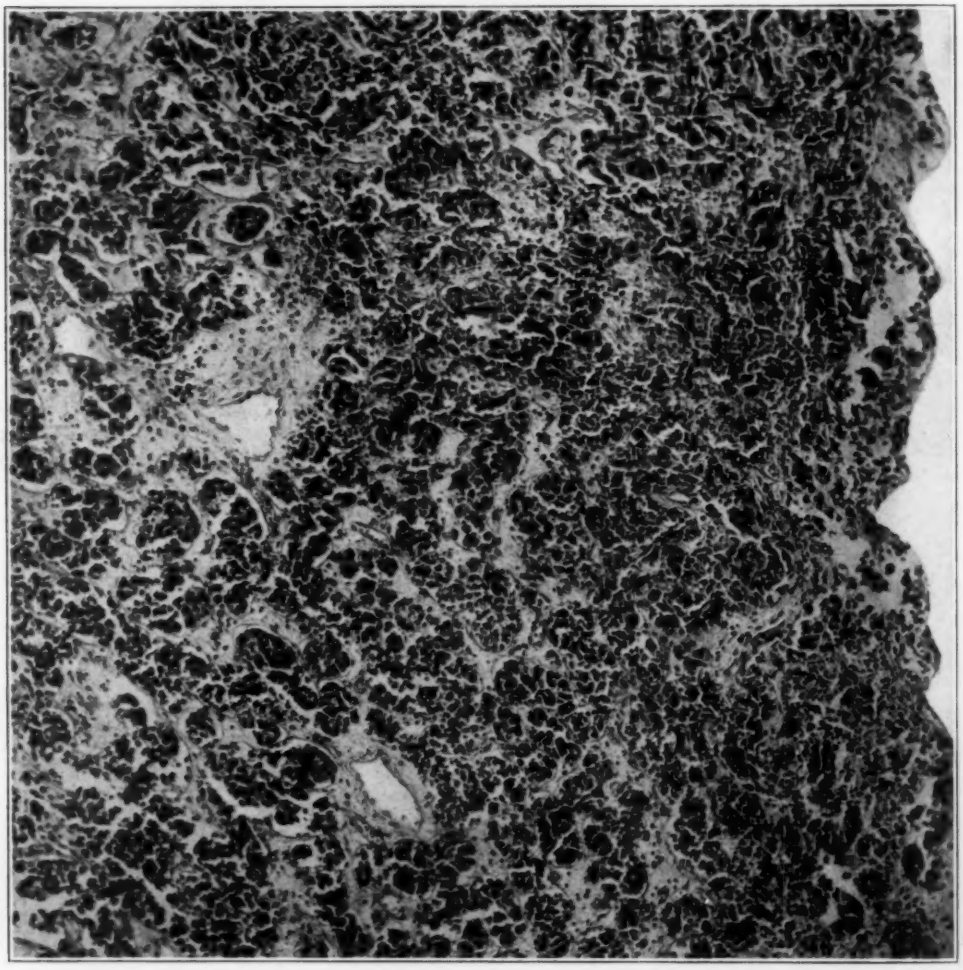

Fig. 3.—General view of the tumor showing its distinct glandular architecture, × 40.

Microscopic Examination of the Tumor.—Sections were taken from the surface as well as from the pedicle of the tumor. On the surface there was occasionally a stratified layer of squamous epithelium (fig. 2); occasionally there was seen a single layer of columnar epithelium. Mostly, however, the surface was denuded of epithelium due to postmortem desquamation. The epithelium rested

on a basal membrane which could be followed up along the entire circumference of the tumor except at the points where the cut section was carried. Beneath the basal membrane there was a rather wide layer composed mainly of elastic fibers. This layer, however, could be seen only in a few places; for the most part the tumor extended up to the surface.

As to the tumor proper, at the first glance one gained the impression of a glandular structure (fig. 2, 3 and 4). This structure was seen throughout all

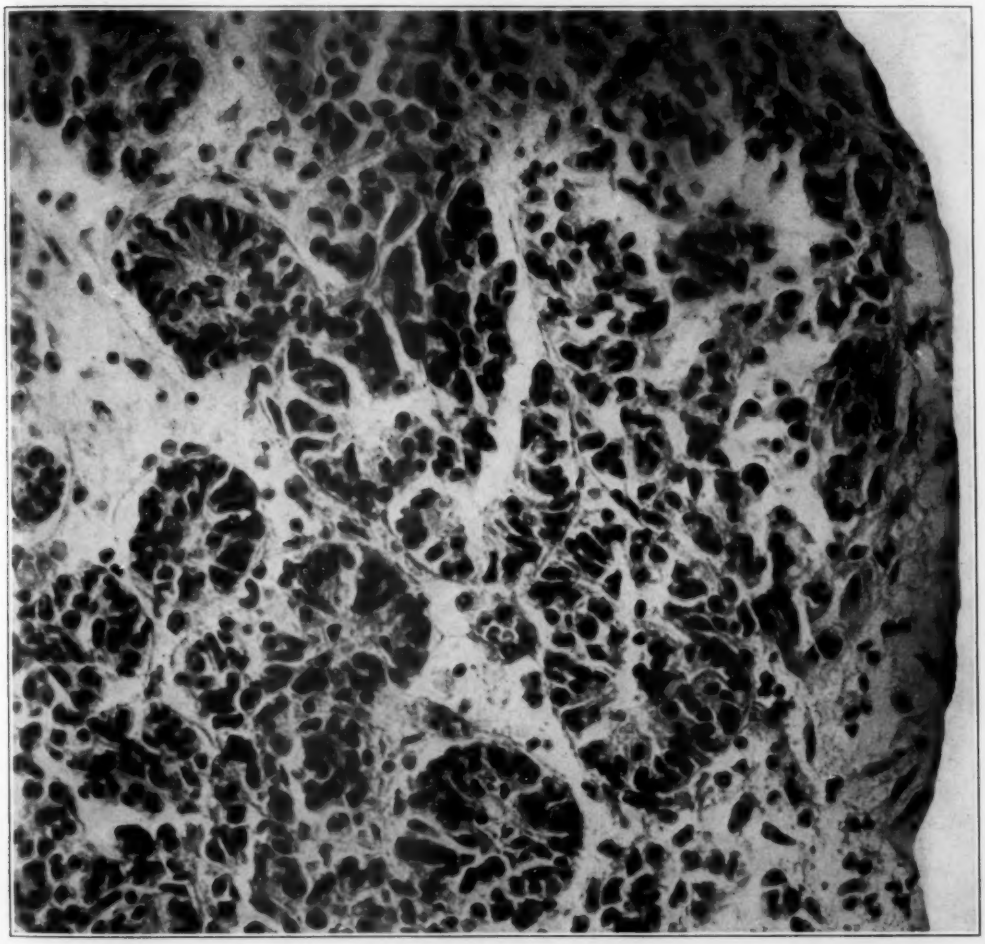

Fig. 4.—High power magnification of the tumor showing the uniform columnar epithelial cells which rest everywhere on a basement membrane; × 200.

the sections. The tumor appeared to be composed of round and oval alveoli often intercommunicating and lined by columnar epithelium which contained a cylindrical rather dark nucleus. The cells rested on a basement membrane (fig. 4). Occasionally, the alveoli contained globular blue stained material, apparently mucus (fig. 5). The stroma was composed of loose connective tissue and showed occasional infiltration with polymorphonuclear leukocytes, plasma cells and lymphocytes and contained numerous thin-walled blood vessels.

On close examination, variations in the structure of the alveoli as well as variations in the cells could be seen distinctly. It was striking that the alveolar structure was more clearly demonstrated in the peripheral portion of the tumor than in the central one. While the cells in the peripheral portion were uniform in appearance, those in the central part differed considerably (fig. 6); they were larger and contained large dark nuclei, the nucleocytoplasm ratio being altered to the disadvantage of the latter. Cells with two and more nuclei and occasional

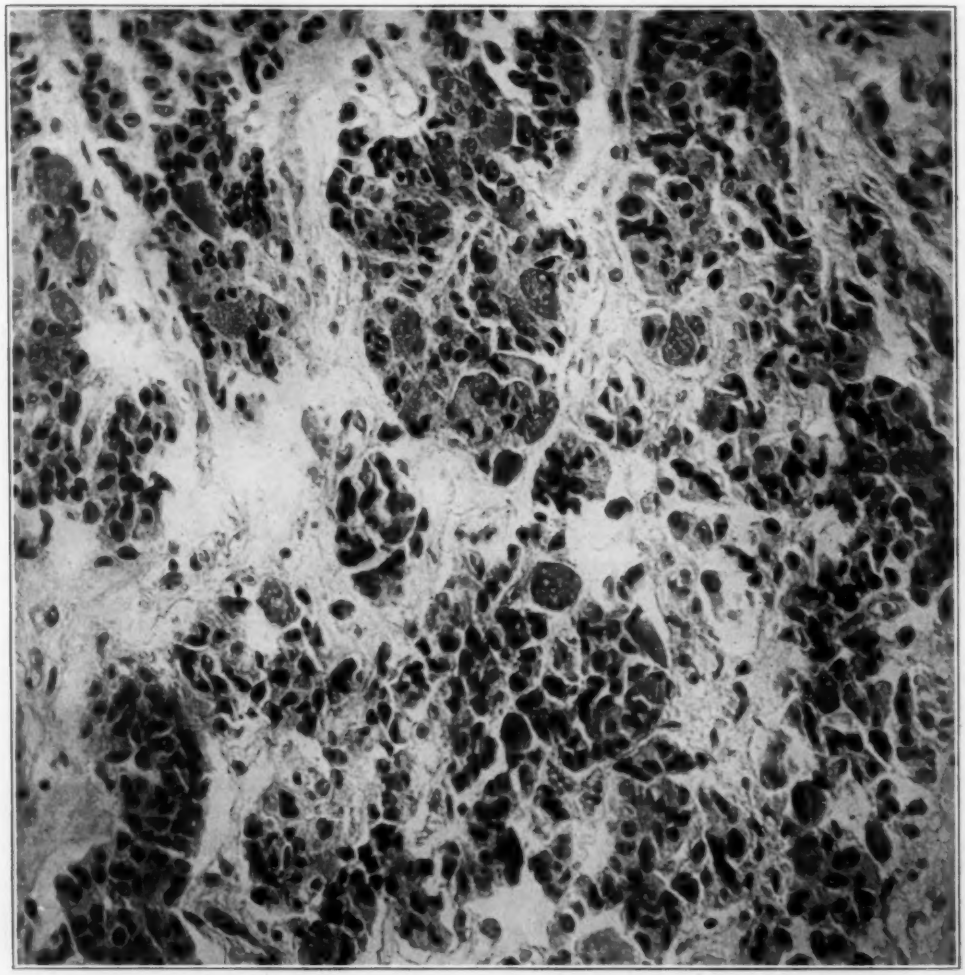

Fig. 5.—A portion of the tumor with mucus secretion within the alveoli; ×160.

mitoses were found. Sections from the pedicle of the tumor did not show any infiltration of the bronchial wall.

From this description it can be concluded that the tumor originated from beneath the bronchial mucous membrane, since it was surrounded in its entire circumference by the basement membrane and the surface epithelium was still

preserved in places. Therefore, it might have originated either from the mucous glands or from their excretory ducts. The columnar shape of the cells composing the alveoli, and the fact that only occasional accumulations of mucus are encountered and the intercommunication of the alveoli, speak in favor of the origin from the excretory ducts.

Grossly, the tumor appeared benign owing to its polypoid shape and its smooth surface; there was neither ulceration of the surface nor any infiltration at the

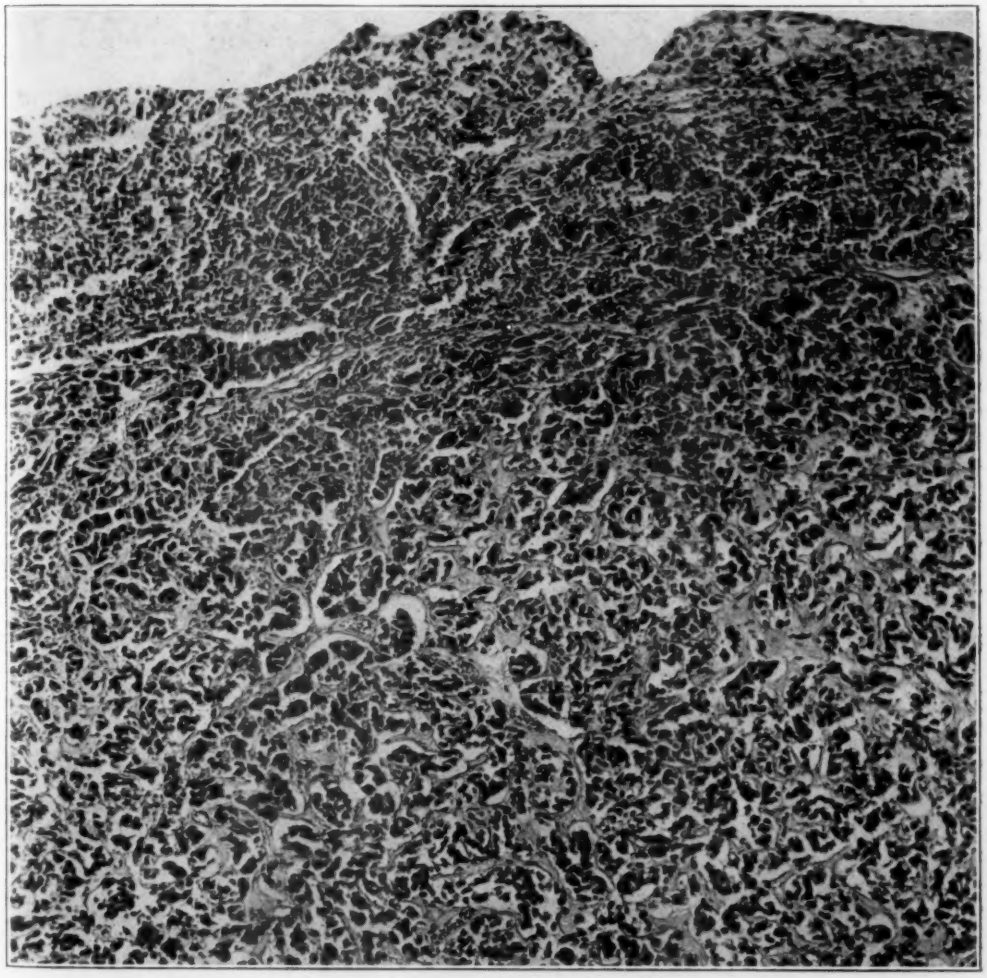

Fig. 6.—An area showing the contrast between the regular structure of the periphery and the polymorphous atypical cells of the central portions; × 40.

base. Microscopically, the presence of the basement membrane supports the gross diagnosis of the benign nature. The deeper portions of the bronchial mucous membrane were compressed, but the basement membrane and the overlying epithelium was still preserved and nowhere had it been perforated. This pointed to an expansive and not an infiltrative growth, which was also in accord with

the mode of growth of a benign tumor. The uniformity in the morphology of the tumor cells forming the alveoli which was found within the greater portion of the tumor was also a point in favor of its benign nature. The distinct morphologic variations in the appearance of the cells in the central portions of the tumor, the alteration of the nucleocytoplasm ratio, the hyperchromatic nuclei and the occasional mitotic figures, however, are features that speak in favor of malignant transformation.

The histologic opinion, therefore, was that of a benign adenoma originating from the epithelium of the excretory ducts of the bronchial mucous glands, but containing portions that exhibited a precipitate proliferation with signs of immaturity and anaplasia.

COMMENT

The case reported here is that of a woman, aged 47, who had suffered for several years from symptoms of a respiratory disease associated with cough and expectoration, and had been repeatedly operated on for empyema in the right side of the chest. Since the symptoms continued she was admitted to the hospital where a cavity in the base of the right lung was found. An operation which was attempted to bring about the collapse of the cavity had to be discontinued on account of a profuse hemorrhage from the wound. After a short period of apparent improvement, another profuse hemorrhage from the wound was followed by almost immediate death. Autopsy revealed a polypoid growth springing off the wall of the main bronchus of the lower lobe of the right lung grossly showing definite benign characteristics, histologically an adenoma with features that make a malignant transformation in the center of the tumor probable. Numerous sacculated bronchiectatic cavities were present within the lower lobe of the right lung distal to the seat of the tumor, one of which opened directly on the surface of the lung and showed a communication with the sinus in the thoracic wall. An erosion of a branch of the pulmonary artery was found within one of the bronchiectases with a terminal hemorrhage from that vessel.

In correlating the postmortem observations with the clinical course of the disease, it appears evident that the symptoms were due to the extensive bronchiectases in the lower lobe of the right lung. The condition diagnosed as empyema was probably the result of the extension of the suppurative process from the bronchiectases into the pleural space; particularly, the cavity opening directly on the surface of the lung can be looked on as a continuous source of maintaining the suppurative process in the thoracic cavity. The final cause of death was the profuse terminal hemorrhage from the eroded branch of the pulmonary artery. It can be said, therefore, that the occluding polypoid tumor in the bronchus of the lower lobe of the right lung which caused the development of the bronchiectases (the mechanism of which will be discussed later) was undoubtedly responsible for the entire course of illness as well as for the final cause of death. The protracted course of the disease,

extending as it did for a period of several years, is consistent with the presence of a benign tumor. The malignant changes revealed by the histologic examination can be regarded as of late occurrence and without significance for the clinical picture and the final events.

REVIEW OF LITERATURE

A survey of the literature in regard to intrabronchial new growth of epithelial origin reveals only a few cases that show features similar to the case here reported. In the case of Mueller,[1] a definitely benign pedunculated adenoma with partial calcification was found in the main bronchus of the left lung causing a partial occlusion of the lumen. Numerous sacculated bronchiectatic cavities filled with purulent and fetid material were present distal to the seat of the tumor. This case was that of a young woman, aged 22, who had suffered for a period of eight years from profuse expectoration of bloody and fetid material. The origin of this tumor was attributed to proliferation of the bronchial mucous glands. Another case of an epithelial polypoid tumor, which was considered as a cylindical cell carcinoma originating from aberrant epithelium, was found by Kreglinger[2] in the main bronchus of the left lung giving rise to almost complete obstruction of the lumen and atelectasis of the lung. In his case there was a history of massive expectorations for a period of six years, and numerous bronchial dilatations could be demonstrated at autopsy. In view of the course of the disease, it seems justifiable to assume that originally this tumor was benign. Kirch[3] reported a case of a polypoid tumor in the main bronchus of the left lung with diffuse sacculated dilatations of the distal bronchi; the histologic opinion was that of an originally benign fibromatous polyp with late malignant proliferation and tendency to infiltrative growth. A polypoid tumor in the main bronchus of the right lung located in close proximity to the lower lobe branch with extensive bronchiectases in the lower lobe of the right lung, was observed by Heck.[4] Malkwitz[5] reported a case of a primary polypoid tumor in the main bronchus of the left lung. Both these cases, however, presented definite malignant features with infiltration of the bronchial wall, and in both cases the origin was attributed to congenitally aberrant epithelium. A true

1. Mueller, Heinrich: Zur Entstehungsgeschichte der Bronchialerweiterungen, Inaug. Diss., Halle a/S 1882.

2. Kreglinger, G.: Ueber ein primaeres Bronchialkarzinom, Frankfurter Ztschr. f. Pathol. **12**:135, 1913.

3. Kirch, Eugen: Ueber stenosierende Bronchialgeschwülste mit konsekutiver Bronchiektasenbildung, Centralbl. f. allg. Pathol. u. pathol. Anat. **28**:545, 1917.

4. Heck, Willi: Ueber primäres polypöses Bronchialkarzinom, Inaug. Diss., Bonn, 1916.

5. Malkwitz, Frieda: Beitrag zur Kenntnis polypöser Bronchialkarzinome, Frankf. Ztschr. f. Pathol. **26**:189, 1922.

polypoid adenoma in the main bronchus of the middle lobe of the right lung with complete obstruction of the lumen and cylindrical bronchiectases of the distal bronchial ramifications has recently been described by Heine;[6] the origin of this tumor is not definitely stated, both the surface epithelium of the bronchus and the epithelium lining the excretory ducts of the bronchial mucous glands are considered as possibilities.

From the foregoing cases, it appears evident that bronchial dilatations were almost invariably encountered in these cases of occluding intrabronchial neoplasms. Indeed, since the thorough investigations of Biermer[7] on the pathogenesis of bronchial dilatations, it is well established that intrabronchial tumors occluding the lumen may give rise to development of extensive dilatations of the distal bronchial ramifications. According to Biermer, the disturbance of the respiratory mechanism leading to an increase in the expiratory intrabronchial pressure is the the factor primarily responsible for the development of the bronchiectases. It is the valvelike action of the occluding growth that affects the expiration much sooner and to a greater extent than the inspiration, which may for a considerable length of time go on undisturbed. The persistent cough driving the air from the surrounding alveoli into the bronchi under forced pressure is again a factor which increases the expiratory intrabronchial tension and which places a strain on the bronchial wall and leads to a gradual weakening of the elastic tissue. The ground for the dilatation appears well prepared and since hypersecretion is usually present in these bronchi, the accumulation of the secretion soon causes inflammatory changes which damage still more the contractile and elastic elements of the bronchial wall, so that finally no force is left that would counteract the dilating factors. If this process goes on for a sufficient length of time the inflammation may spread to the parenchyma of the lung, and as a result of this the normal elasticity of the lung is diminished or lost. As the lung is then not able to follow sufficiently the expansion of the chest during inspiration, an increased traction is exerted on it by the increased inspiratory negative pressure. This ultimately may act as an extrabronchial factor favoring the dilation of the bronchi.

Biermer's conception of the mechanism and the pathogenesis of bronchiestases distal from the point of bronchial occlusion seems to be well illustrated by the type of tumors with which we are dealing here. The well circumscribed pedunculated tumor endowed with more or less mobility appears particularly fit to reproduce the mechanism with its valvelike action. This is especially true for the slow growing benign

6. Heine, J.: Ueber eine primäre gestielte Bronchialgeschwulst, Verhandl. der deutschen pathol. Gesell. **22**:293, 1927.

7. Biermer: Zur Theorie und Anatomie der Bronchienerweiterung, Virchows Arch. f. path. anat. **19**:94, 1860; ibid., **19**:241, 1860.

tumor without tendency to infiltration, but with a decided occluding growth which may exist for a considerable length of time as a silent tumor without causing symptoms of great significance. The cases in which only partial occlusion of the bronchus existed seem to illustrate this mechanism much clearer than the cases of complete bronchial obstruction. If a bronchus has been completely occluded at a relatively early period of the existence of the tumor, the possibilities for the development of bronchiectases become slight, since the particular portion of the lung supplied by this bronchus loses its function completely and becomes atelectatic.

It is, therefore, the tendency to gradual occlusion of the lumen and the noninfiltrative growth that explains the fact that benign tumors in the bronchi are usually found associated with extensive bronchiectases. In addition to the foregoing cases of epithelial tumors there have been recorded several cases of benign occluding intrabronchial neoplasms of nonepithelial origin. A case is mentioned by Rokitansky [8] in which a lipoma in the main bronchus of the lower lobe of the left lung obstructed the lumen almost completely; the distal bronchial branches were dilated and the lower lobe of the left lung collapsed. Radestock [9] reported a rather unusual case of an obstructing tumor of the main bronchus of the right lung with numerous bronchial dilatations and a cavity opening directly on the surface of the lung. Histologically, the tumor proved to be composed of thyroid tissue and its origin attributed to an aberrant thyroid. Siegert [10] reported a case of a chondroma within a branch of the bronchus of middle lobe of the right lung and Blecher [11] a case of a pedunculated ossified chondroma in the main bronchus of the left lung near the bifurcation; both of these cases presented numerous cylindrical bronchiectases distal from the seat of the tumor. A case of a pedunculated fibroma in the bronchus of the lower lobe of the right lung with cylindrical bronchiectases was described by Knack.[12] A lipomalike tumor in a branch of the bronchus of the middle lobe of the right lung was reported by Feller.[13]

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8. Rokitansky, Carl: Lehrbuch der pathologischen Anatomie, 1861, vol. 3, p. 25.

9. Radestock: Ein Fall von Struma intratrachealis, Beitr. z pathol. Anat. **3**:289, 1888.

10. Siegert: Ueber primäre Geschwülste der unteren Luftwege, Virchows Arch. f. path. anat. **129**:413, 1892.

11. Blecher: Ueber die klinische Bedeutung der Bronchialekchondrosen, Mitt. a. d. Grenzgeb. d. Med. Chir. **21**:837, 1910.

12. Knack: Ein Fall von Bronchiektasenbildung infolge eines Fibroms an der Abgangsstelle des unteren Hauptbronchus, Deutsche med. Wchnschr. **44**:1007, 1918.

13. Feller, Adolf: Ueber ein lipomähnliches Hamartom der Lunge, Virchows Arch. f. path. Anat. **236**:470, 1922.

Those rare cases of tumor development in cases of bronchiectases of long standing, such as those reported by Chiari,[14] must be regarded as a different entity. In a case of diffuse chronic tuberculosis of both lungs with occasional sacculated bronchiectases, he observed a tumor in one of the cavities which histologically proved to be a proliferating adenoma originating from the bronchial mucous glands. The other case of Chiari was that of a mixed tumor, a lipochondro-adenoma in an old sacculated bronchiectatic cavity. These tumors are to be considered as secondary to the bronchiectases being the sequel and not the cause of the latter. Such tumors, according to Chiari, may develop in the course of the dilatation of the bronchi in a similar manner as proliferation of the papillary type commonly found in old bronchiectases.

The fact that extensive bronchiectases are less frequently found in cases of malignant bronchial tumors is explained by their more rapid growth, the metastases and the general deleterious effect on the organism which usually cause death long before there is sufficient time to develop bronchiectases. The ulceration and the degenerative changes which these tumors frequently undergo may also be a factor in preventing a marked degree of bronchial occlusion. Occasionally, however, marked bronchiectases are seen in malignant neoplasms, particularly in the nodular or papillary form as in the cases of Biermer,[7] Muethler [15] and Reiche.[16]

We mentioned at the outset the importance of bronchoscopy as a valuable diagnostic aid in intrabronchial neoplasm. Reports in the literature indicate that there is also a therapeutic field for this method, particularly in cases of benign tumors. Spiess [17] removed a polypoid tumor of the main bronchus of the right lung lodged just at the bifurcation by means of the bronchoscope after tracheotomy had been performed. The patient who had suffered from severe dyspnea with attacks of suffocation showed marked improvement. An endothelioma of the main bronchus of the right lung was removed by Jackson [18] by means of peroral bronchoscopy and the patient observed for one and one-half years as free from symptoms. Pfeiffer [19] used the same method in

14. Chiari: Zur Kenntnis der Bronchialgeschwülste, Prag. med. Wchnschr. **8**:497, 1883.

15. Muethler, Gustav: Ein Fall von Bronchostenose durch ein Sarkom bedingt, Inaug. Dissertation, Berlin, 1873.

16. Reiche, F.: Primäres Tracheakarzinom, Metastase in der linken Nebenniere, Melasma suprarenale, Centralbl. f. allg. Pathol. u. pathol. Anat. **4**:1, 1893.

17. Spiess, Gustav: Ein Fall von hochgradiger Dyspnoe infolge eines Polypen im rechten Bronchus, München. med. Wchnschr. no. 40, p. 2095, 1910.

18. Jackson, Chevalier: Endothelioma of the Right Bronchus Removed by Peroral Bronchoscopy, Am. J. Med. Sc. **153**:371 (March) 1917.

19. Pfeiffer, Willy: Das Jacobson-Holzknecht'sche Phänomen bei einseitiger Bronchostenose und seine künstliche Erzeugung, Deutsche med. Wchnschr. no. 47, p. 1298, 1920.

removing a fibroma from the main bronchus of the left lung and has seen the patient well seven years after the operation. Another case of fibroma in the main bronchus of the left lung is mentioned by Wessler and Jaches;[20] on roentgen-ray examination this case showed an almost complete atelectasis of the left lung, which after bronchoscopic removal of the tumor cleared up rapidly. Recently Orton[21] reported a bronchoscopic removal of a pedunculated growth from the bronchus of the middle lobe of the right lung; although in this case the histologic opinion was in favor of a malignant tumor, the patient was well four years after the removal of the growth.

We have seen that intrabronchial tumors, while being benign both in their gross appearance and histologically, may lead to rather serious and even grave consequences due to their particular location. An early diagnosis and operative procedure, especially removal by means of bronchoscopy may often prevent extensive pathologic changes and be of great benefit for the patient.

SUMMARY

1. A case of a polypoid adenoma of a large bronchus originating from the excretory duct of the bronchial mucous glands is described.

2. The mechanism of the consecutive formation of bronchiectases is discussed.

3. The literature on the subject is reviewed.

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20. Wessler and Jaches: Clinical Roentgenology of Diseases of the Chest, Troy, N. Y., 1923, p. 51.

21. Orton, Henry Boylan: Carcinoma of the Bronchus, S. Clin. N. Amer. **6**:1534, 1927.

DISSECTIONS OF THE NECK*

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Observations of the too frequent indifferent methods and the correspondingly poor results obtained in dissections of the neck in general, invite an attempt to develop an orderly and clearcut technic and a definite understanding of the objects to be attained. The substance of this paper recalls the teaching of the surgical masters of the past and present and, in addition, records the experiences of the authors throughout a decade of surgical operations of the neck.

CLINICAL INDICATIONS

Dissections of the neck are performed for metastatic carcinomas, primary endotheliomas, localized Hodgkin's disease, intractable tuberculosis and for less frequent conditions, such as lymphosarcoma, carcinoma of the brachial cleft and tumors of the carotid body. Our purpose is to outline a method for the eradication of malignant processes which are primary elsewhere and metastatic in the neck. Primary carcinoma of the lower lip or of the skin of the face below the mouth metastasizes to the glands immediately inferior to the mandible, while carcinoma of the cheek, tongue, tonsil, pharynx, antrum and the lateral portions of the face, metastasize primarily to the lateral regions of the neck. Accordingly, from the surgical metastatic standpoint, the neck is divided into two metastatic regions, i. e., the suprahyoid region for carcinoma about the lip and the lateral region of the neck for the other growths enumerated.

ANATOMIC CONSIDERATIONS

The structures of the neck are so complex and the surgical procedure is so fraught with dangers in the hands of the uninformed, that a thorough anatomic understanding of the neck is of first importance.

A. Suprahyoid Region of the Neck.—The suprahyoid region (fig. 1) or, in general, that region between the mandible or lower jaw and the hyoid bone, is divisible into three compartments: a submental compartment and two submaxillary compartments.

The submental division of the suprahyoid region is a triangle bounded laterally by the anterior bellies of the digastric muscles and inferiorly by the hyoid bone. The apex of this isosceles triangle is at the symphysis of the mandible. The anterior wall or covering of the submental

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compartment is made up of the skin and the platysma muscle as well as a sheet of enveloping or deep fascia of the neck. Its posterior wall or floor is composed of the anterior portions of both mylohyoid muscles. The contents of this compartment are simply loose areolar tissue containing lymph glands without important nerves or blood vessels.

Each submaxillary region is also a triangle bounded laterally and below by the two bellies of the digastric muscle, and above by the horizontal ramus of the mandible prolonged to the parotid gland. It is covered by the skin and platysma, deep to which is attached a thin layer

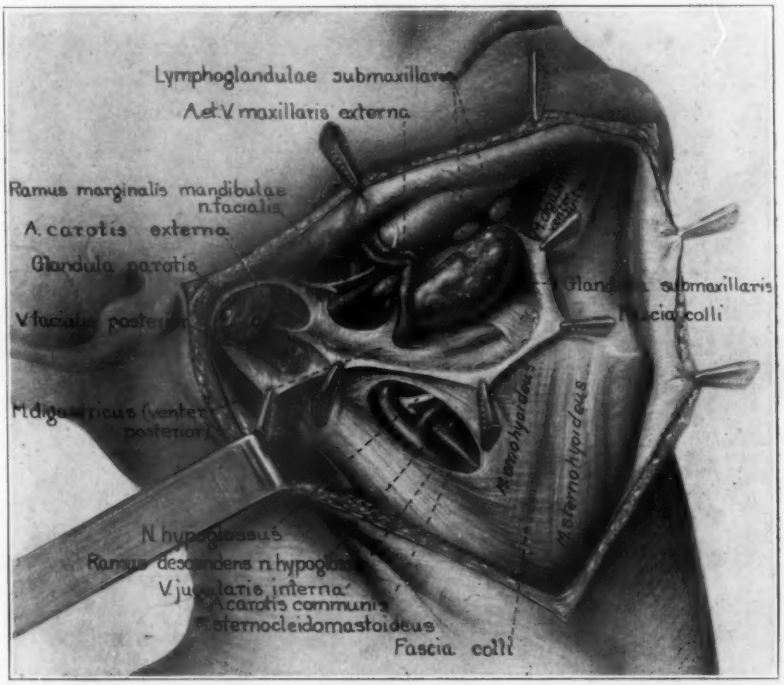

Fig. 1.—Dissection to show the submaxillary compartment. The skin-platysma flaps expose the enveloping fascia of the neck which has been partly removed to show the contents of the compartment and its investment of the parotid gland together with its relation to the great vessels.

of enveloping aponeurosis continuous with that over the submental triangle. The floor or back wall is composed of the lateral portions of the mylohyoid muscle and the underlying hyoglossus muscle projected laterally to the anterior border of the sternoleidomastoid muscle and the parotid gland.

The contents of the compartment are more important than those of the submental triangle. Each contains a submaxillary salivary gland, the facial vessels, a quantity of loose areolar tissue in which are

embedded several lymph glands, and the more vital structures, namely, the hypoglossal and the lingual nerves. The facial vessels traverse the compartment in its lateral half and cross over the horizontal ramus of the mandible at a point approximately midway between the angle and the symphysis. The hypoglossal nerve crosses the compartment near its inferior angle where the digastric bellies attach themselves to the hyoid bone. It lies deep to the mylohyoid muscle, and its further course over the hyoglossus is in the cleft between the two muscles. The lingual nerve lies hidden by the ramus of the jaw and courses along and in the sulcus between the under surface of the mandible and the floor of the hyoglossus. Perhaps the largest branch leaves the nerve at the posterior margin of the mylohyoid and immediately enters the substance of the submaxillary gland.

Two important near-related structures which do not actually lie within the compartment are the marginal mandibular branch of the facial nerve and the inferior pole of the parotid salivary gland. The marginal mandibular nerve courses along the outer surface of the mandible, deep to the platysma, but superficial to the enveloping fascia. The inferior pole of the parotid gland is separated from the true compartment by only a thin fascia and is thoroughly exposed in both anatomic and clinical dissections.

B. Lateral Region of the Neck.—Under the heading of the lateral region (figs. 2 and 3) of the neck we include all the structures between the trapezius muscle laterally and the ribbon muscles of the thyroid gland mesially, the clavicle inferiorly, and the posterior belly of the digastric muscle with the base of the skull superiorly. This region so described includes, therefore, the rest of the neck, only those structures which are strictly infrahyoid being omitted, i. e., the thyroid gland, the laryngotracheal tube and the esophagus.

The roof or outer covering of this general region is made up of the skin platysma layer and the enveloping layer of the deep fascia of the neck. The posterior wall or floor is the prevertebral fascia covering the deep muscles of the neck. This space is naturally divided into subcompartments, i. e., a region overlaid by the sternocleidomastoid muscle, the the sternocleidomastoid or carotid region, and the region posterior to it, the posterior cervical triangle.

(*a*) The Carotid or Sternocleidomastoid Region: This region is an elongated quadrilateral space, bounded by the borders of the sternocleidomastoid muscle, by the mastoid process and the clavicle. In depth, it is considered to extend to the prevertebral fascia.

The sternocleidomastoid muscle, invested by the splitting of the aforementioned enveloping fascia which helps form the roof of the entire neck, may be said to overlie the region at hand. In consideration of this muscle, it should be here noted that the spinal accessory nerve

enters and leaves it in its superior third. Deep to this roof muscle is the omohyoid muscle, an important landmark. The pretracheal fascia, or the anterior prolongation of the enveloping aponeurosis of the neck, forms here a more or less distinct layer, being somewhat cellular and areolar above the omohyoid muscle and resistant to a greater degree including and inferior to it. Its importance lies in the fact that it furnishes a fibrous expansion from the clavicle to the hyoid bone which acts more or less as a protection to the great vessels.

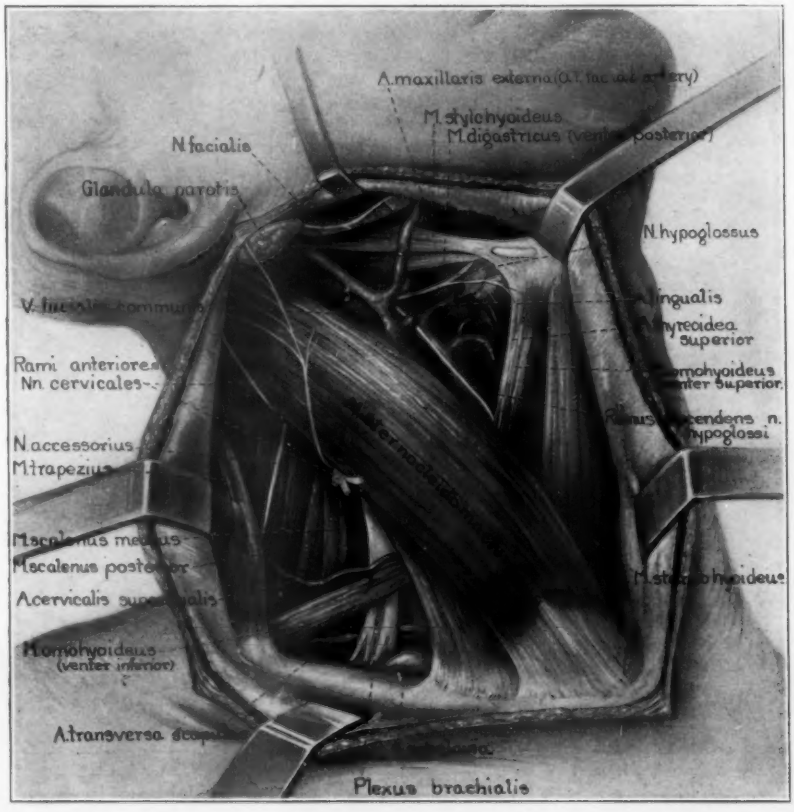

Fig. 2.—The lateral region of the neck. The skin-platysma flaps are turned back and the enveloping fascia colli is removed. The drawing is designed to show the sternocleidomastoid or carotid and the posterior cervical divisions of the lateral neck with the sternocleidomastoid in situ. Attention is called to the highly vascular superior extremity of the carotid region.

The contents of this retromuscular compartment include chains of lymph glands embedded in areolar tissue, the great vessels of the neck and the vagus nerve enclosed within the carotid sheath, the thyrolingual-facial veins, the hypoglossal nerve and on the left side of the neck, the thoracic duct.

The lymph glands are largely those of the superior deep cervical chain, and they lie mainly in the loose areolar tissue about the vessels in the upper two thirds of the region. A few glands of this chain extend a little below the junction of the two bellies of the omohyoid muscle, and a few supraclavicular glands of the subclavian or inferior deep cervical chain lie along the upper margin of the clavicle. There is continuity between the superior deep cervical chain of glands and

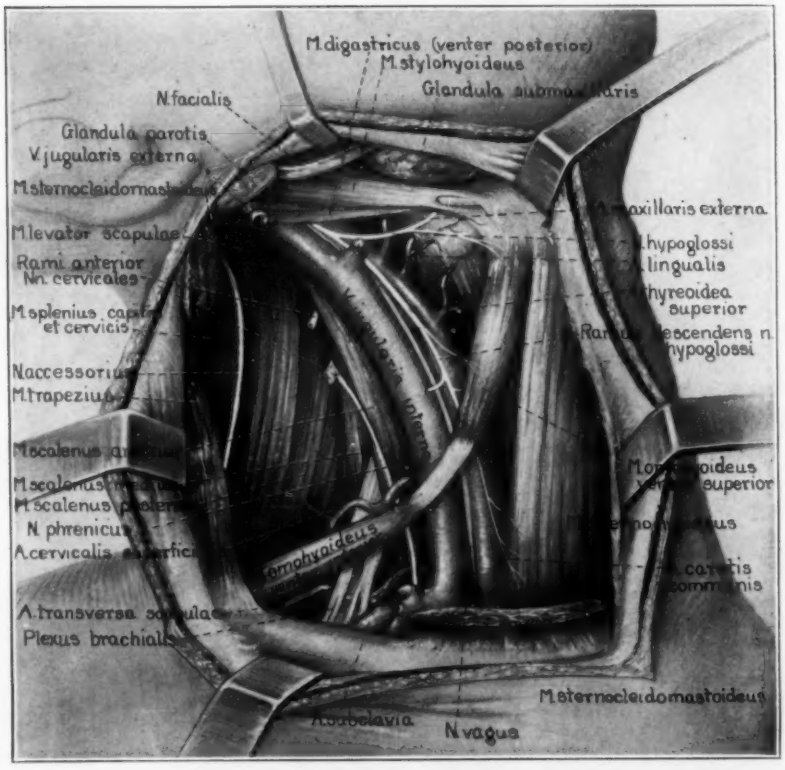

Fig. 3.—The lateral region of the neck. The sternocleidomastoid muscle is removed to show the structures deep to it and the continuity of the carotid and the posterior cervical divisions of the region.

the submaxillary glands superiorly and with the inferior deep cervical or supraclavicular glands inferiorly.

The contents of the carotid sheath are the common carotid artery, the internal jugular vein and the vagus nerve. The common carotid artery runs from below upward under cover of the mesial border of the sternocleidomastoid muscle as far as the upper margin of the thyroid cartilage, where it bifurcates into its internal and external branches. Over the course of the common carotid artery the internal jugular vein

lies external to it. Superior to its bifurcation, however, the internal carotid artery runs posterior to the internal jugular vein in its course to the base of the skull. There are no branches to the internal or the common carotid artery in the neck, while the external carotid in our region gives off the superior thyroid, lingual and external maxillary branches. The superior thyroid artery leaves the parent trunk about 1 cm. from the bifurcation and the lingual and facial vessels at a more distant point.

The internal jugular vein begins at the base of the skull deep and anterior to the mastoid process of the temporal bone and deep to the external auditory meatus. It passes downward and forward beneath the sternocleidomastoid muscle and lateral to the common carotid artery.

The vagus nerve leaves the skull beside the internal jugular vein and courses mesial to this vein as far as the common carotid artery, where it lies between and behind these structures in the carotid sheath until its entrance into the mediastinum.

The hypoglossal nerve emerges from the base of the skull near the two foregoing structures and courses with them for a short distance to a point on a level with the hyoid bone, so that it runs forward and upward and crosses between the posterior belly of the digastric muscle and the hyoglossus muscle to the submaxillary compartment, as has been already described. The spinal accessory nerve leaves the skull soon to traverse the superior third of the sternocleidomastoid muscle and to be distributed through the posterior compartment to the trapezius muscle.

The veins from the face, the tongue and the thyroid gland enter into a more or less common trunk, the thyrolingual-facial trunk, which lies in the superior portion of the region and communicates with the internal and external jugular vein.

The anterior branches of the cervical plexus, with the exception of the phrenic nerve, pierce the prevertebral facia to emerge through the posterior triangle at about the posterior margin of the sternocleidomastoid muscle. There are the great auricular and the lesser occipital nerves and, in general, the nerves that supply the scapula. The phrenic nerve lies on the anterior scalene muscle deep to the prevertebral fascia or the floor of the region.

The parotid gland, while encased in a compartment of its own is, nevertheless, in intimate relation not only with the submaxillary region, but with the superior portion of the sternocleidomastoid region. Its importance in this discussion lies in the fact that some of the lymph glands of the superior deep cervical chain are in close contact with its inferior pole.

Within the region on the left side the thoracic duct enters this compartment from the chest and empties into the junction of the left subclavian and the left internal jugular vein. The cords of the brachial

plexus can be seen beneath the floor of the region through the transparent prevertebral fascia and do not in any way belong in this space.

(*b*) The Posterior Compartment or the Posterior Cervical Triangle: The posterior compartment is a triangle bounded posteriorly by the trapezius muscle, anteriorly by the sternocleidomastoid muscle, and inferiorly by the clavicle. Its floor or posterior wall is made up of the prevertebral fascia, a continuation of the floor of the sternocleidomastoid region. The areolar lymph gland bearing tissue, which, for the most part, makes up the contents of this compartment comprises glands of the superior deep cervical chain as well as inferior deep cervical systems draining the axilla and mediastinum. The glands of these two deep

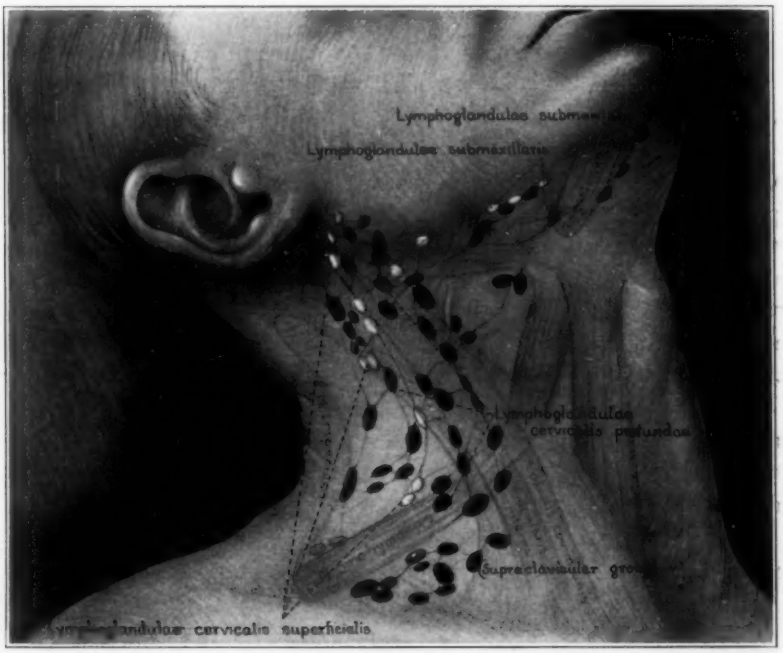

Fig. 4.—Schematic drawing to represent the distribution of the lymphatic gland chains of the neck and their relationship to one another and the neighboring structures. The deeper glands of the cervical chain are the ones primarily involved in carcinoma of the tongue. Note the close relationship between these glands and the internal jugular vein; the highest glands lying against the vein at the base of the skull and embedded in the lower pole of the parotid gland; the lowest gland at the lower edge of the omohyoid muscle as it crosses the vein, and the barren zone between this group and the suprahyoid chains constituting a break between the deep cervical glands and the supraclavical chains. The suprahyoid chains are primarily involved in carcinoma of the lip. The glands first affected are usually those lying against the ramus of the jaw along the edge of the submaxillary salivary gland and those at the apex of the submental triangle just beneath the point of the chin.

cervical chains are separated by the posterior belly of the omohyoid muscle. The only important other structure in that area of the compartment superior to the omohyoid muscle is the spinal accessory nerve. This nerve runs a variable course from the posterior edge of the sternocleidomastoid muscle to the anterior border of the trapezius. The important structures in the area inferior to the omohyoid muscles are the subclavian artery and vein and several large branches of the acromiothoracic trunk. Other vitally important structures of the neck are not occupants of this compartment, i. e., the cords of the brachial plexus as they emerge from the interval between the scalenus anticus and the scalenus medius muscle, the phrenic nerve, the inferior thyroid artery and the sympathetic chain, for these structures lie deep to the prevertebral fascia and, therefore, beneath the floor of the region.

SURGICAL DISSECTION OF THE SUPRAHYOID REGION

Indication.—The suprahyoid dissection, independent of the dissection of the lateral region of the neck, is used for metastatic carcinoma of the lip when the indications for the complete dissection of the neck are lacking; that is, when gross examination of the submental and submaxillary glands, at operation, do not show evidence of metastases. A complete or a combined dissection of the neck is necessary when there is gross involvement of the submaxillary or submental groups. This follows from the general rule regarding metastatic carcinoma that secondary chains must be removed where there is gross involvement of the primary chains. Seldom, however, does carcinoma of the lip require more than the simple operation on the suprahyoid region.

The uncombined, or the pure suprahyoid dissection of the neck is often a bilateral procedure, because usually the lesion of the lip crosses the midline or is near the midline. The bilateral dissection adds little to the operation, and it gives the patient a symmetric neck. This procedure should follow the resection of the local primary lesion as soon as convenient, and always within a few weeks, providing the microscopic study of the primary lesion shows a growth of sufficient extent to have infiltrated the muscle of the lip or in any case regardless of the extent of the growth where there is a total lack of differentiation of cell (Broeders).

Method of Procedure.—A "cut throat" incision is employed (fig. 5), extending from mastoid process to mastoid process, and passing across the front portion of the throat just above the hyoid bone. This incision is modified if a unilateral suprahyoid dissection alone is contemplated, and then extends from the mastoid process only a short distance across the midline to the anterior belly of the opposite digastricus at its insertion on the cornu of the hyoid bone.

The superior flap is dissected up, taking platysma with skin and subcutaneous tissue, while the inferior flap is undisturbed. The upper flap is turned back to the insertion of the platysma or until the lower portion of the outer surface of

the mandible is exposed. This lays bare the contents of the triangles to be dissected, covered by the enveloping fascia. With a sharp blade this fascia is divided all along the anterior border of the mandible and over the parotid gland from chin to sternocleidomastoid muscle. By a "wiping down" process or blunt dissection, the separation of the glandular and areolar contents from the periosteum of the bone, from the parotid gland, and from the outer surface of the mylohyoid and hyoglossus muscles is accomplished. This cleaning-out process is early held up by the facial vessels, which must be ligated and cut where they pass over the horizontal ramus of the jaw (fig. 6). At this point, one proceeds with caution until the marginal branch of the mandibular nerve (seventh) is isolated and retracted out of danger. This nerve can be located best and traced out by picking it up as it comes across the ramus of the jaw together with the facial vessels (Maxillaris externa). The destruction of this nerve means a permanent paralysis of the lower angle of the mouth. The "wiping down" process may now continue with the cleaning off of the outer surfaces of the

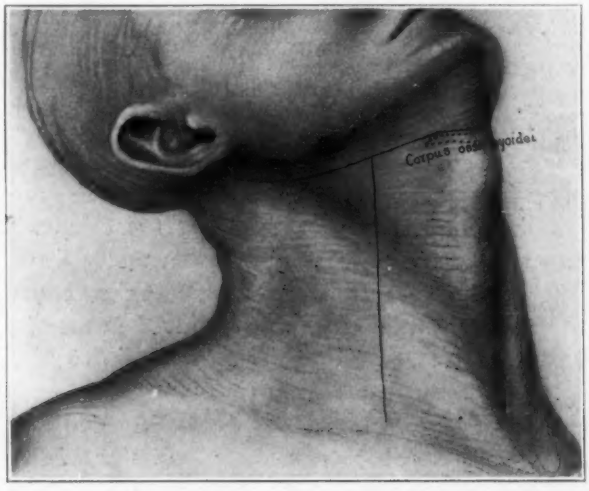

Fig. 5.—Incisions employed for dissections of the neck. The horizontal line represents the incision for suprahyoid unilateral dissection. It extends from the mastoid to the anterior belly of the digastricus of the opposite side. It lies in a natural fold of the neck and passes just above the hyoid bone. Deviation from this line should not be made regardless of possible wrinkles, as this always gives the best cosmetic result. The upper flap only is lifted. The vertical line represents the incision for the lateral dissection. It extends from the middle of the horizontal incision and strikes the clavicle just lateral to the sternocleidomastoid muscle at its insertion on that bone.

mylohyoid and hyoglossus muscle. It is again halted by the submaxillary branch of the lingual nerve, by the submaxillary duct and by the plexus of veins about the duct. The submaxillary branch of the lingual nerve enters the superior pole of the salivary gland; accordingly, traction downward of this gland, along with the areolar and lymph glandular contents of the triangle, pulls the lingual nerve into view (fig. 7). The junction point of the nerve branch with the lingual nerve is situated just beneath the lateral border of the mylohyoid muscle near its upper edge. Retraction medialward of the lateral border of the muscle is

usually necessary for exposure. Division of this salivary branch frees the gland and allows the lingual nerve to retract out of danger. The salivary duct is exposed where it enters the base of the tongue by strong traction medialward on the lateral edge of the mylohyoid muscle. Ligations and divisions of the duct and veins release the last hold on the triangle contents. Again caution needs to be observed because the hypoglossal nerve traverses the triangle near the salivary duct and sometimes in the midst of the mass of veins. Before ligation of these veins in this angle, therefore, the nerve should be identified and pushed aside. The dissection is now completed without further interruptions by the final clearing off of the mylohyoid and the hyoglossus muscles down to the posterior belly of the digastricus and the styloid muscles and the central

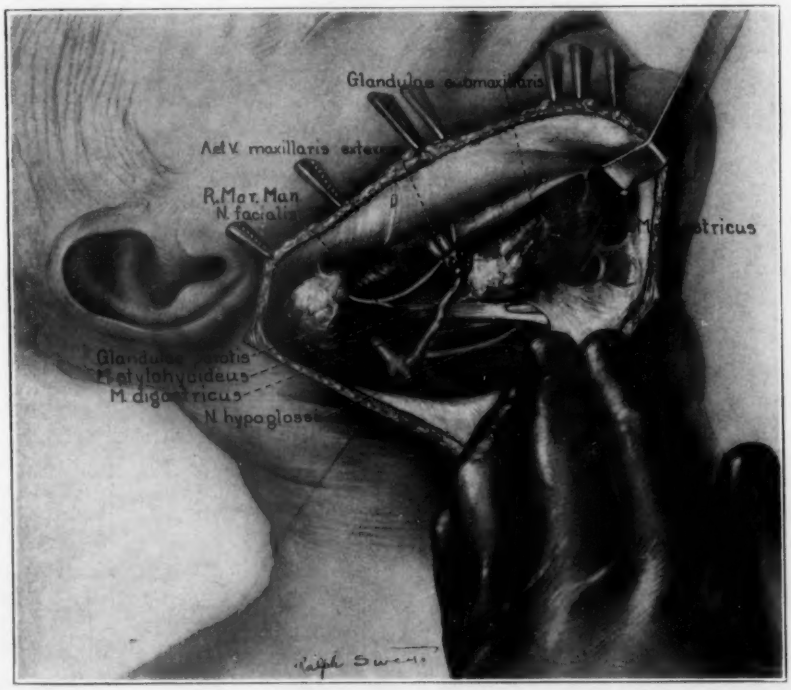

Fig. 6.—Early stage suprahyoid dissection. The facial vessels have been divided, the marginal branch of the mandibular (seventh) has been isolated and preserved. The branch of this nerve to the platysma muscle must be divided and is so pictured. The lower pole of the parotid gland has been cleaned off and is still intact. The submaxillary salivary gland has been freed from the ramus of the jaw by the sharp division of the cervical fascia. The drawing is not a true representation of the actual dissection in that the submental triangle is not shown and the lower flap has been lifted to show structures below the posterior digastric belly. The submental triangle is emptied in all dissections of the neck, while the lower flap in the pure suprahyoid dissection is never lifted.

ligation of the facial vessels. The facial vein crosses over the digastricus muscle, while the artery comes up from beneath that muscle.

The tissues removed should include the submaxillary salivary gland, the facial vessels where they cross the space, and all the lymph glands and loose areolar

tissue. The contents of the submental region, while removed separate from those of the submaxillary compartment, are, in reality, continuous with those of the submaxillary and lie on a common floor. The anterior belly of the digastricus muscle, therefore, must be lifted so as to thoroughly clean the mylohyoid floor. The parotid gland is not disturbed in this dissection.

SURGICAL DISSECTION OF THE LATERAL REGION OF THE NECK

Indications.—Dissection of the lateral region of the neck is sometimes carried out just as subsequently described, but usually it is combined with and follows the suprahyoid dissection. The combined suprahyoid and lateral operation, with or without modifications, is performed for metastatic carcinoma of the buccal cavity, for primary carcinoma of

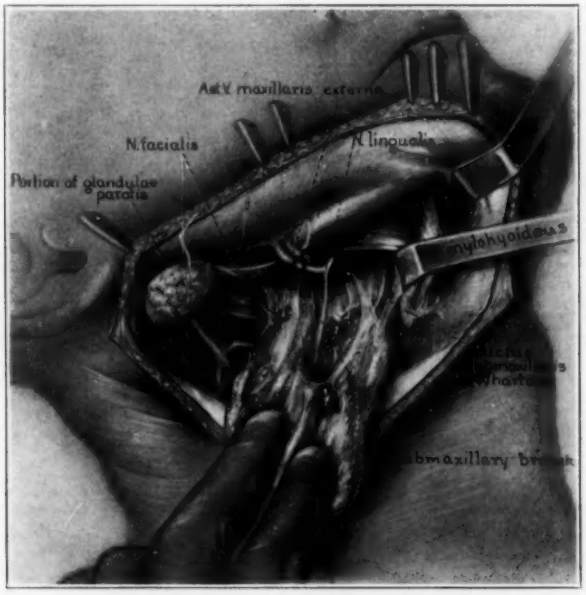

Fig. 7.—A late stage in the suprahyoid dissection. The lingual nerve has been pulled down into view. It will retract out of danger when the branch to the submaxillary salivary gland has been divided. Wharton's duct is about ready for ligature and division. The hypoglossal nerve crosses the angle made by the two bellies of the digastricus muscles just beneath the duct on the hyoglossus muscle floor at the point represented by the star.

the branchial cleft, with or without metastases, for primary endothelioma arising in the lymph glands of the neck, for Hodgkin disease and occasionally, for severe cervical tuberculosis. It is carried out without modifications in all instances of metastatic carcinoma and must be performed in all cases of primary carcinoma within the buccal cavity regardless of metastasis. This holds true regardless of the size of the lesion or the extent of the infiltration with the possible exception of

occasional instances of early and strictly localized carcinoma of the cheek or of the gums. Dissection of the neck may precede or follow, or may be combined with the operation on the local lesion. Usually it should precede this operation so as to block off the local area to be treated.

Method of Procedure.—The incision for the combined suprahyoid and lateral dissection is T-shaped, the horizontal incision being simply that already described for the unilateral suprahyoid dissection (fig. 5). The vertical incision is dropped from the midpoint of this horizontal line and strikes the clavicle near the lateral edge of the sternocleidomastoid muscle. This same incision is employed where the lateral dissection alone is to be made.

The two lateral flaps consisting of skin, fat and platysma are turned back, the posterior as far as the anterior edge of the trapezius muscle and the anterior so as to expose the ribbon muscle of the thyroid (fig. 8). The area to be cleaned out is then outlined posteriorly by the division of the enveloping fascia along the anterior edge of the trapezius muscle and the clearing away of the areolar tissue in this plane down to the prevertebral fascia covering the floor of the neck. This exposes the spinal accessory nerve and divides many sensory branches of the cervical plexus. The area is outlined medially by the division of the enveloping and pretracheal fascia just lateral to the ribbon muscles of the thyroid, with the consequent exposure of the common carotid artery (fig. 8). The inferior limits of the area to be dissected are worked out as follows: The sternocleidomastoid muscle is cut transversely about 5 cm. above the clavicle, or approximately at the level of the crossing of the emohyoid muscle. The clavicular stump of this muscle is cleaned off on its under surface and turned back down over the clavicle. It is used later to fill the dead space in the supraclavicular fossa. By blunt and sharp dissection, the supraclavicular tissue is now divided along a straight line paralleling and about 3 cm. above the clavicle or at a level approximately 2 cm. below the omohyoid where it crosses the deep vessels. This line must be below the tendinous middle portion of the omohyoid in order to include the whole of the deep cervical chain of lymph glands because the lowest gland in this chain lies just below the omohyoid muscle over the lateral wall of the internal jugular vein (fig. 4). It need not be placed lower than this line because the glands about the subclavian vessels need not be removed even when the lowest deep cervical gland is grossly involved, clinical experience having shown that the subclavian chain is rarely involved in instances of metastatic carcinoma of the neck even when that condition is advanced. Also at this level the operation is more simple, owing to the fact that there is no danger of wounding the subclavian vessels. As the tissues are divided the first important structure seen is the external jugular vein, which is ligated and divided at the lateral corner. Almost at the same depth at the medial angle the internal jugular vein is exposed. This vessel is also picked up, doubly ligated and divided. The dissection is now carried down to the floor of the neck, that is, to the prevertebral fascia overlying the deep muscles. Many small vessels must be ligated and some nerves may be cut, but no structures of any vital importance are encountered.

With the accomplishment of this inferior delimitation the isolation of the contents of the lateral region of the neck is now complete except at the deep superior extremity, and the actual removal may be commenced. The dissection should start from below and proceed upward, following the floor of the neck, that is, following the outer surface of the prevertebral fascia. This process is one of turning upward of the areolar gland-bearing tissues and with them the

internal jugular vein (fig. 9). In this step the common carotid artery, the vagus nerve and the prevertebral fascia are thoroughly cleaned of loose areolar tissue. The upward dissection may be carried out without fear of mishap or danger to any vital structures because the vagus nerve and common carotid artery are always plainly visible, and the brachial plexus and phrenic nerve are protected by the prevertebral fascia. This fascia, which is never disturbed, is identified as a transparent tough covering closely adherent to the deep muscles of the neck. All branches of the cervical plexus come out through this fascia

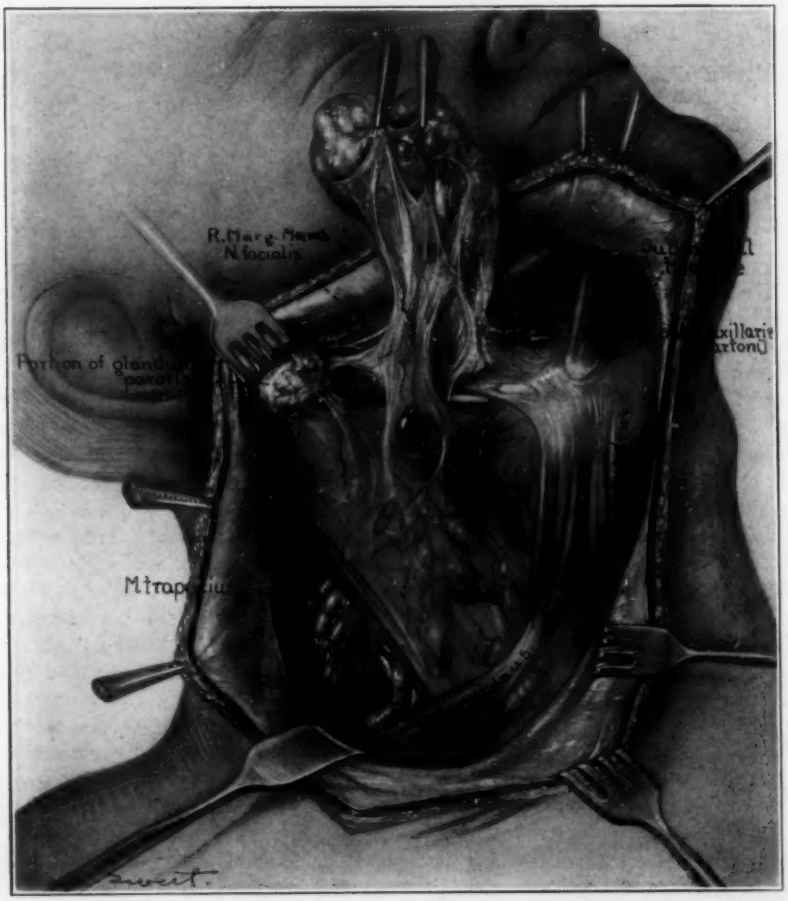

Fig. 8.—Early stage of the complete dissection of the neck. The suprahyoid dissection has been completed down to the upper margin of the posterior belly of the digastricus. The lateral flaps of skin and platysma have been dissected back. The edge of the trapezius marks the lateral limits and the thyroid muscles mark the medial limits of the area to be dissected. The lower limits are defined by dividing the omohyoid and sternocleidomastoid muscles at the level of the crossing of the lower edge of the omohyoid and the lateral edge of the sternocleidomastoid.

except the phrenic nerve, and all, with this one exception, are divided at their point of emergency, through the fascia. No branches of the carotid artery will be seen until the bifurcation which takes place at about the level of the most prominent part of the "Adam's apple." Usually the first indication that the point of bifurcation has been reached is the finding of a branch, the superior thyroid, apparently coming off from the common carotid vessel. This is due to the fact that the internal carotid lies behind the external at the bifurcation and both are enclosed with a common sheath for one or more centimeters. The bifurcation can always be demonstrated, however, as taking place proximal to this

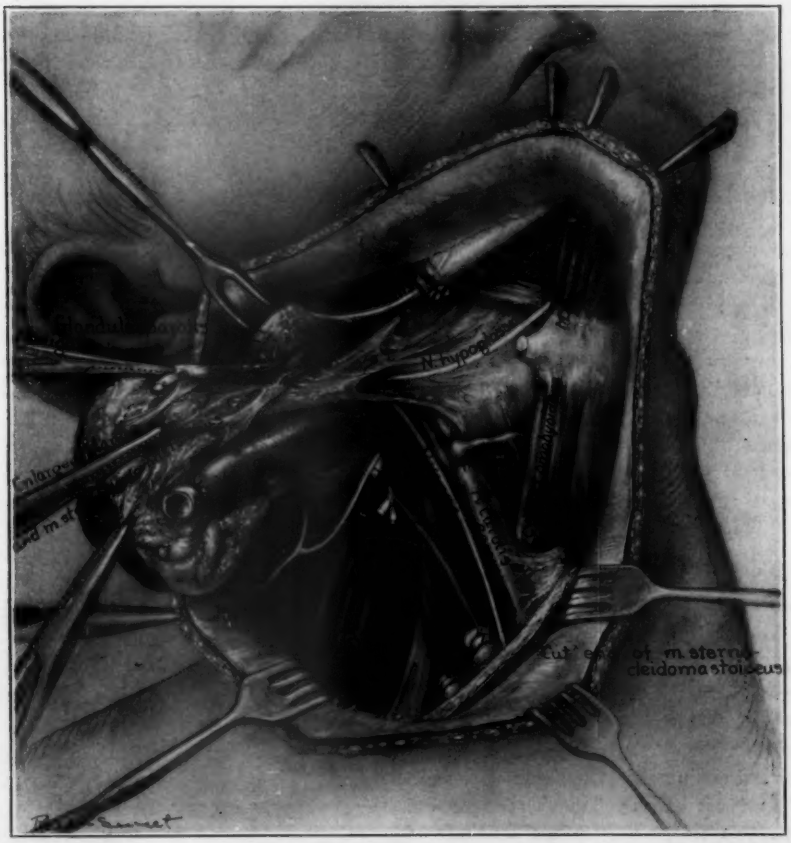

Fig. 9.—Later stage of complete dissection of the neck. The area to be dissected has been outlined below by the division of the sternocleidomastoid and omohyoid muscles, the ligature and division of the external and internal jugular veins, and the separation of the areolar tissues down to the prevertebral fascia or floor of the neck. The divided structures have been turned upward in the process of clearing off the floor; the carotid artery and vagus nerve have been stripped of all loose tissue, the branches of the cervical plexus except the phrenic have been cut as they emerge through the prevertebral fascia. The phrenic nerve lies unharmed behind the protecting layer of prevertebral fascia. The posterior belly of the digastricus muscle is shown divided, cleaned off and retracted. It is sutured later and continues to function. It is taken only when involved in a carcinomatous mass.

branch. As soon as the identification is clear, a ligature is slipped about the external carotid artery and tied just proximal to the superior hyoid branch, thus insuring the ligature from slipping off the end. Both vessels are then divided after clamping or ligating the distal stumps. The upward dissection should now proceed smoothly providing the medial area is dissected first until the tendinous portions of the stylohoid and digastricus muscles and the hypoglossal nerve are exposed. Great care must be taken to isolate the hypoglossal nerve which makes a loop below the level of the posterior belly of the digastricus muscles, extending frequently 2 cm. below the lower border at the central portion of the belly and crossing under the muscle in its tendinous portion at the hyoid cornu (fig. 9). The nerve is easily identified rather deep on the floor of the neck if sought for beneath the hyoid cornu. Dissection may now continue upward and lateralward following along the stylohyoid muscle in the direction of the styloid process, and is usually carried through to the base of the skull. Frequently, however, it is not possible to proceed further than the isolation of the hypoglossal nerve because of metastatic masses which prevent the proper turning up of the contents of the neck and the complete exposure of the floor of the neck at the base of the skull. Working in this corner without sufficient room may result in the nicking of the internal jugular vein on its under surface with an obscuring of the field and discoloring of the tissues from flooding of the area with venous blood.

The last step takes place at the upper lateral angle, and has to do with the cutting away of the sternocleidomastoid muscle from the mastoid process, with the taking away of the lower pole of the parotid gland, and the ligation of the internal jugular vein at the skull (fig. 10). The lower part of the parotid gland (approximately from 1 to 2 cm.) must be taken because some of the lymph glands at the upper end of the deep cervical chain lie embedded within this structure. In cutting through the parotid, innumerable veins, many of considerable size, must be divided, and progress here usually is slow. On completing this dissection the most difficult task still remains, i.e., the isolation and ligation of the internal jugular vein and the identification and preservation of the vagus, the hypoglossal and the spinal accessory nerves. These nerves lie close to the floor of the neck, also close to the vein, so that mass ligature is not possible. It is well to approach the vein from one side and then the other, leaving the direct working down on the vessel as the last step. The complete removal of the internal jugular vein is required because of two facts, namely, the intimate relationship of the lymphatic vessels and glands with the sheath of the vein all along its course and the location of the highest gland in the deep cervical chain against the vein at the skull (fig. 4). The spinal accessory nerve too often must be sacrificed in the radical dissection for carcinoma, because in its passage through the sternocleidomastoid muscle it frequently becomes involved in a tumor mass and cannot be separated without danger of leaving carcinomatous tissue behind.

With the completion of the dissection, the floor of the neck is clear; that is, the prevertebral fascia has been cleaned of all loose areolar tissue and is everywhere intact. The vagus, hypoglossal, and frequently, the spinal accessory cranial nerves may be seen lying on the floor of the neck, the vagus lying against the lateral wall of the common carotid artery, the hypoglossal looping down beneath the posterior belly of the digastricus muscle and disappearing underneath its end at the hyoid bone, and the spinal accessory passing obliquely downward and backward to disappear beneath the edge of the trapezius near the lower angle of the wound at the lateral corner. The common and internal carotid arteries are

also seen traversing the floor of the neck from the clavicle to the base of the skull. A row of stumps of branches of the cervical plexus penetrating the prevertebral fascia, that is, the floor of the neck, lies just lateral to the artery. The phrenic nerve may be seen coming out from behind the lateral edge of the scalenus anticus muscle together with the nerve to the lowest stump, but instead of piercing the fascia and suffering division with the other branches of the cervical plexus, it courses down along the anterior surface of the muscle belly beneath the prevertebral fascia and disappears behind the clavicle into the chest. The brachial plexus also is seen beneath the same fascia at the extreme lower and

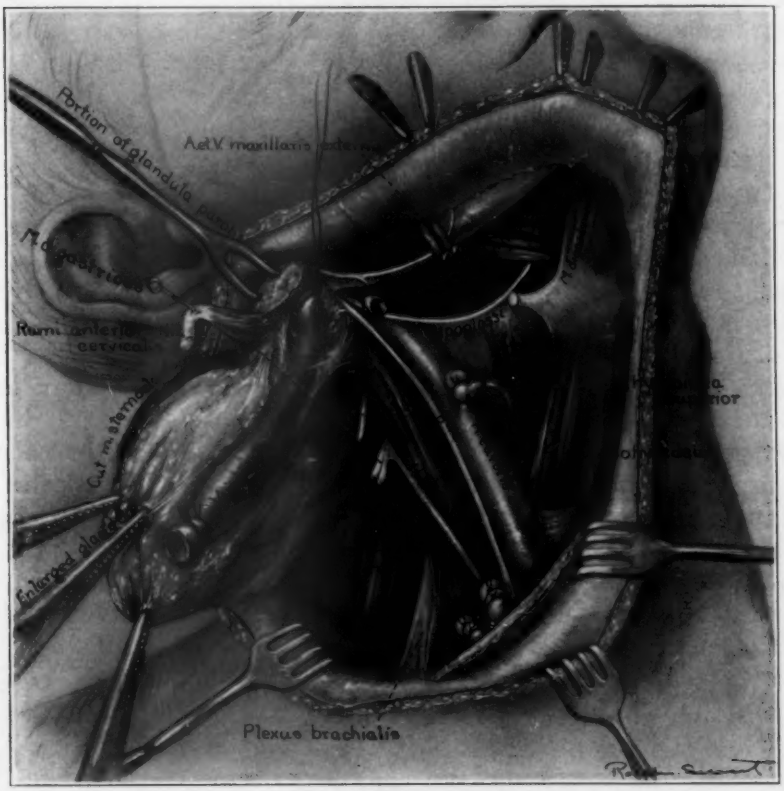

Fig. 10.—Last stages of complete dissection of the neck. The external carotid artery has been doubly ligated and divided. The hypoglossal nerve has been stripped and carefully preserved. The lower pole of the parotid gland has been taken away and the jugular vein has been ligated at the base of the skull. This cannot be shown accurately because the point of ligation would be too high up under the parotid and angle of the jaw to be visible from this angle. The vagus, hypoglossal and accessory nerves lie close to the vein at the site of ligature and must be identified before the ligature is placed.

medial angle alongside and lateral to the common carotid artery. The remaining mass of loose areolar tissue surrounding the subclavian vein and covering the apex of the lung, pumps up and down with respiration.

COMMENT

Modifications of the combined suprahyoid and lateral dissections of the neck may be allowed in tuberculosis and Hodgkin's disease. In such cases the sternocleidomastoid muscle may be preserved, the cervical nerves are spared, and the internal jugular vein is taken only as a matter of convenience. In metastatic carcinoma, in carcinoma of the branchial cleft and in endothelioma of the neck the procedure is never more limited than that outlined in the foregoing description of the combined operation. Sometimes additional structures must be removed; occasionally the hypoglossal nerve and the digastricus and stylohyoid muscles must be dissected out because of their involvement within a large carcinomatous mass. Even the vagus nerve sometimes must be sacrificed. Removal of less than the whole of the deep cervical chain in metastatic carcinoma involving these groups of glands is just as inadequate as a simple amputation without axillary dissection for carcinoma of the breast.

Simple suprahyoid dissection is an absolute safeguard in cancer of the lip. It should be employed early in the course of the disease before gross involvement of glands has taken place, and should not be delayed until there is little doubt regarding the malignant nature of the swelling or enlargement of the glands beneath the chin. Microscopic examination of the glands may fail to reveal metastatic carcinoma in a surprising number of instances, but statistical studies show that in a high percentage of the cases in which dissection of the neck is not employed the patients return to the physician later on account of metastases. This is especially true of the cases which show involvement of the muscle of the lip or in which there is a slight degree of differentiation of cells. Dissection of the neck in the suprahyoid region is not mutilating; no vital structures are in danger, and the procedure is simple and can be accomplished by one with a limited surgical experience; on the other hand, the results from the standpoint of protection are absolute.

The combined suprahyoid and lateral dissection for metastatic carcinoma in the deep cervical chains is almost as satisfactory even in the presence of gross involvement of these glands, and in the majority of instances gives a complete and permanent block to the spread of the disease by metastases. While it is true that cures are relatively few in carcinoma of the tongue or the pharynx, it is the inability to get rid of the primary focus and the infiltration of the floor of the mouth or pharynx and not the inability to control metastases that accounts for the failure. The later dissection, while not as simple as the suprahyoid, can be easily mastered, and is not a formidable procedure for surgeons who are skilled in handling tissues, even though they have had little experience in operations on the neck. Even in this extensive procedure, mutilation is negligible. The bilateral combined operation is seldom employed,

but may be performed without fear of serious consequences. It is wise to allow sufficient time between the two operations for the establishment of collateral circulation.

Apropos of recent discussions concerning the anesthetic to be employed in complete dissection of the neck, it has been our experience that the most satisfactory anesthetic is light ether, that is, a minimum of ether and a maximum of air. Local anesthesia with procaine hydrochloride, nitrous oxide and oxygen, ether and chloroform have been employed. Ether is preferred because postoperative pneumonia and mortality have been negligible and because the patients have been more comfortable following this type of general anesthetic. With local anesthesia the discomfort of lying on the table throughout the long period required for a painstaking operation of this sort is a point to be weighed. There is more oozing under nitrous oxide because of the unavoidable cyanosis, be it ever so slight; in addition, the method of administration is more difficult because of the proximity of the operative field to the face. Obstetric chloroform would be the ideal anesthetic in the hands of an anesthetist absolutely familiar with the drug.

Ether should be administered intrapharyngeally with some machine adapted to furnish air under positive pressure. The intratracheal method of administering ether anesthesia is to be avoided because of the high mortality when this method is used, because of the difficulty of administration, and because it is not an improvement on the intrapharyngeal method.

USE OF INTRA-ARTERIAL INJECTIONS OF SODIUM IODIDE IN DETERMINING CONDITION OF CIRCULATION IN THE EXTREMITIES

REPORT OF CASES *

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Impairment of the circulation in the extremities due to diseased blood vessels, which results in gangrene and the symptoms accompanying it, is of ever growing importance. The conditions responsible for these failures of circulation may be considered as aneurysms of various types and obstructions to the arteries and veins. The acute obstructions may be due to emboli or thrombi obstructing important vessels, the result of which may be gangrene if either the arterial or venous blocking is sufficiently complete. The more chronic types of gangrene are the result of arterial disease, and clinically and pathologically, these are recognized as: (1) the arteriosclerotic type which embraces senile and diabetic gangrene, (2) thrombo-angiitis obliterans (Buerger's disease) and (3) arterial spasm (Raynaud's disease).

Each of these three types of disease of the blood vessels differs distinctly in its pathologic changes. In the arteriosclerotic type, the entire arterial system is involved, the capillaries and arterioles being seriously involved, and the development of a compensatory collateral circulation is hardly possible. In thrombo-angiitis obliterans, the capillaries and arterioles are not affected to a corresponding extent, and the development or improvement in the collateral circulation is possible. In Raynaud's disease, an arterial spasm exists rather than permanent changes in the blood vessels. This knowledge should be kept in mind, and efforts at treatment of patients with gangrene or impending gangrene of the extremities should be guided accordingly.

In the arteriosclerotic type, in spite of many reports to the contrary, arterial sympathectomy is probably not indicated. If methods other than palliative measures or amputation are used, obstruction of the venous return of the blood from the extremity should at least theoretically be resorted to by partially blocking the femoral vein and thereby utilizing to the fullest extent the diminished amount of blood which passes through the small arteries. It may be that in some cases the good results which have followed arterial sympathectomy or the injecting of alcohol around the artery for the same purpose, as practiced by

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* Read before the Southern Surgical Association, Augusta, Ga., December, 1927.

Sampson Handley,[1] in senile gangrene, have been due to the partial obstruction of the femoral vein by the swelling of the surrounding tissue following the operative procedures.

In the second type of gangrene (Buerger's disease), there is a possibility of an improvement of the collateral circulation, and with the lower extremity involved, the ligation of the femoral artery as practiced by Dr. Dean Lewis [2] would possibly be the treatment of choice.

The uniformity of the arterial channels and their collateral circulation can usually be relied on to guide one to the most favorable site for amputation. But because one cannot tell the exact condition of the vessels, one may sacrifice an unnecessary length of leg, or again one may amputate at too low a level, so that the condition for which amputation was performed would not be relieved. In order to determine more definitely the location of the vascular obstruction, many methods have been suggested, no one of which has been entirely satisfactory. The Mosskowicz method is well known, and the test depends on the alteration of the color of the skin which results from the alternate application and removal of a tourniquet. But this method is often inadequate and unreliable.

The change in the temperature of the foot or toe produced by the application and removal of a tourniquet, as recommended by Brooks [3] following some excellent observations, is more promising and with development may be of great value. The apparatus consists of a thermocouple mounted in a hypodermic needle and a galvanometer. It permits of accurate measurements of the temperature of the foot and records accurately rapid change in temperature. The tourniquet is applied for ten minutes, and the temperature of the tissues of the extremity falls to room temperature. It was found that with the removal of the tourniquet the temperature in the tissues of the normal extremity rapidly rose to normal. On the other hand, in the tissues of an extremity in which there was impairment of the arterial blood supply, the release of the tourniquet was not followed by a prompt rise in temperature.

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1. Handley, W. Sampson: Treatment of Gangrene of the Extremities, Proc. Roy. Soc. Med. **20**:485 (Feb.) 1927.

2. Lewis, Dean: Spontaneous Gangrene of the Extremities, Arch. Surg. **15**:613 (Oct.) 1927; Lesions of the Blood Vessels of the Extremities, South. M. J. **20**:421 (June) 1927.

3. Brooks, Barney: Intra-Arterial Injection of Sodium Iodide, J. A. M. A. **82**:1016 (March) 1924; Diseases of the Blood Vascular System of the Extremities, J. Bone & Joint Surg. **6**:326 (April) 1924; New Method for Study of Diseases of the Circulation of the Extremities, **7**:316 (April) 1925. Brooks and Jostes: A Clinical Study of Diseases of the Circulation of the Extremities, Arch. Surg. **9**:485 (Nov.) 1924.

Still another method of determining the condition of the circulation in the extremities is by the injection of sodium iodide into the arteries and by roentgenologic examination. Brooks is also credited with making and publishing important experimental and clinical observations in the use of this method. He recommends the exposure of the femoral vein and artery in Scorpa's triangle, and with the vein and artery clamped above, 10 cc. of 100 per cent sodium iodide is injected into the artery with a syringe and needle and a roentgen-ray exposure is rapidly made on a sensitive plate. The pain is severe, and a short gas anesthesia is required. I have made use of this method in six patients since the publication of Brooks' work. The technic employed has been slightly different in that a light spinal anesthesia was used in four patients; this seemed to have the advantage of securing the cooperation of the patient. It was also not found necessary to expose the vessels by an incision, but the solution was easily injected with a needle through the skin, while an assistant exerted pressure over the artery and vein just below the inguinal ligament. The results of my experiences cause me to both recommend and to condemn the method. It is because of the unfavorable results, as much as the favorable, that I am prompted to make a record of my observations.

Three of the six cases that are reported, occurred in patients with aneurysms: one of the popliteal artery, one an arteriovenous aneurysm of the femorals and one an aneurysm of the first part of the brachial artery. The results in these three cases were satisfactory, and the information gained was invaluable. The other three cases occurred in patients with gangrene of the toes or feet. Two were of the senile or arteriosclerotic type, and the other was a moist gangrene due to venous thrombosis of the femoral vein associated with sepsis. In these cases, the results were decidedly unfavorable. While the arterial tree showed perfectly and the condition of the circulation was revealed thoroughly, the damage to the already diseased and narrowed arterioles and capillaries from the sodium iodide was evident; in two instances, the gangrene spread rapidly, necessitating a high amputation in one, while death followed in the other.

REPORT OF CASES

CASE 1.—E. S., a colored man, aged 30, entered the hospital on Nov. 8, 1922, because of chronic ulcers of the leg which would not heal. Physical examination at this time revealed the classic signs of an aneurysmal ligament.

The diagnosis was chronic syphilitic ulcers of the left leg and arteriovenous aneurysm of the left femoral artery and vein. Healing of the ulcers occurred following the application of dressings and antisyphilic treatment. The patient left the hospital on December 11, with the ulcers improved, but the condition of the aneurysm was the same. The patient reentered the hospital on Jan. 16, 1923, with ulcers of the left ankle and poor circulation in the left leg. Results of physical examination at this time were essentially the same as those on the

first admission. Roentgen-ray examination of the arterial tree showed the connection of the varix to be a wide communication between the artery and vein. Operation confirmed the diagnosis of an aneurysmal varix, and the afferent and efferent arteries were ligated and divided. The patient recovered from the operation, and the ulcers about the ankles healed rapidly. When the patient was dismissed from the hospital on March 16, the circulation in the leg and foot was fairly good. A low localized bruit could still be heard over the middle third of the thigh. The patient reentered the hospital on June 30, because of fracture of the base of the skull. He recovered completely from this condition. On July 11, the femoral artery was again injected with 10 cc. of 100 per cent sodum iodide, and a roentgenogram was made which showed the ligated ends of the artery, the profunda femoris and a large number of small collaterals connecting the two portions of the artery. The patient was discharged from the hospital on July 25, as improved.

The information gained by the arterial injection previous to the operation was of value in that it showed the inadvisability of attempting to separate the artery and vein and suture the separate openings, which method probably would otherwise have been attempted.

CASE 2.—D. P., a colored man, aged 46, was admitted to the hospital on July 19, 1924, his chief trouble being granuloma inguinale which had produced a urethral fistula. He had also complained of pain in the left leg which caused him to limp or to have difficulty in walking because of a "knot" at the posterior part of the joint of the knee. A diagnosis of popliteal aneurysm was made. Twenty cubic centimeters of 100 per cent sodium iodide was injected into the femoral artery, and a roentgenogram was made. This showed an enormously dilated femoral artery of the left leg with a popliteal aneurysm with partly calcified walls. The condition of the entire femoral artery as shown by injection of iodine indicated that operative treatment was not advisable.

Without the roentgen-ray observations, I think unquestionably that an intrasacular suture of the aneurysm would have been attempted; this I am sure would have resulted in failure, and most likely in gangrene of the leg.

CASE 3.—A. A., a colored man, aged 39, entered the hospital on Aug. 17, 1927, suffering from gunshot wounds of the chest and arm. One of the bullets had penetrated the right axillary region and had produced a huge hematoma in the right axilla. There was also considerable swelling in the region of the right shoulder. The immediate swelling subsided, and about one month after admission a small swelling was noticed in the right axilla just at the junction of the axillary and brachial arteries. This swelling gradually increased in size and became pulsating. About six weeks after admission, the swelling was about the size of a lemon and showed all the classic signs of an aneurysm. Eight cubic centimeters of 100 per cent sodium iodide was injected directly into the aneurysm, and a roentgenogram was made which showed that the aneurysmal sac and the arteries below it were well injected. Immediately after the injection, the extremity showed some paleness, which rapidly disappeared without detrimental effects. One week later the patient was operated on, and a metal clip was applied around the third part of the axillary artery and tightened until the radial pulse was practically obliterated. At the present time, the aneurysm has almost disappeared, and the circulation of the extremity shows signs of slight venous obstruction.

The profunda artery was here revealed, leaving the brachial immediately adjacent to the sac of the aneurysm. It was found so intimately associated with the sac that the contemplated matas intrasacular suturing was abandoned

Figure 1

Figure 2

Fig. 1 (case 1).—Arterial tree after ligation of the artery above and below the aneurysm. The roentgenogram of intra-arterial injection of arteriovenous aneurysm made before operation was lost.

Fig. 2 (case 2).—Intra-arterial injection of popliteal aneurysm showing enormously dilated femoral artery above with aneurysmal sac in popliteal space.

because of fear of destroying the profunda. If the profunda had been obstructed, the entire collateral circulation, which was between the profunda and the circumflex vessels, would have been cut off; this would have resulted in gangrene of the arm.

CASE 4.—Mrs. J. C., white, aged 28, who entered the hospital on April 10, 1925, gave a history of having been delivered of a postmature, full term, dead fetus four weeks previously. Following this she had chills, fever and exquisite pain on the slightest pressure over the middle of Poupart's ligament. All the symptoms were aggravated; the leg became markedly swollen, and the foot began to turn back. A diagnosis of moist gangrene from thrombosis of the left femoral and iliac vessels was made. About six weeks later, a line of demarcation had been established at the ankle joint. At this time, 15 cc. of 100 per cent sodium iodide was injected into the femoral artery, and a roentgenogram was made which showed that the arteries of the leg were normal. There was

Fig. 3 (case 3).—Intra-arterial injection showing aneurysm of first part of brachial artery revealing profunda branch coming off close to the aneurysmal sac.

some blanching of the healthy tissue, and the gangrene which had become stationary immediately spread up the leg several inches. Two months later the leg was amputated at the seat of election. Primary union occurred, and the patient was discharged from the hospital on September 15 in a good condition.

The deleterious effect was evidently due to the irritation of the vessels by the solution, because the venous obstruction caused the drug to be retained overly long before it was returned to the general circulation.

CASE 5.—A. P., a colored woman, aged 60, entered the hospital on Feb. 24, 1925, with a condition diagnosed as senile gangrene of the right foot, which extended up to the midtarsal joint. It was the usual dry type. After six weeks of expectant treatment, sodium iodide was injected into the femoral artery. Following the injection the leg became cold, and the circulation below the knee was seriously affected. Two days later, the patient died.

The patient was old, and her general condition unfavorable; however, her death was apparently hastened by the injection.

CASE 6.—B. F. S., a white man, aged 86, entered the hospital on Feb. 4, 1926, with a history of having been hurt in an automobile accident. Physical examination at this time showed a fairly well nourished man with abrasions on the right side of the chest and tenderness there, but roentgen-ray examination did not show fracture. While in the hospital the patient complained of a

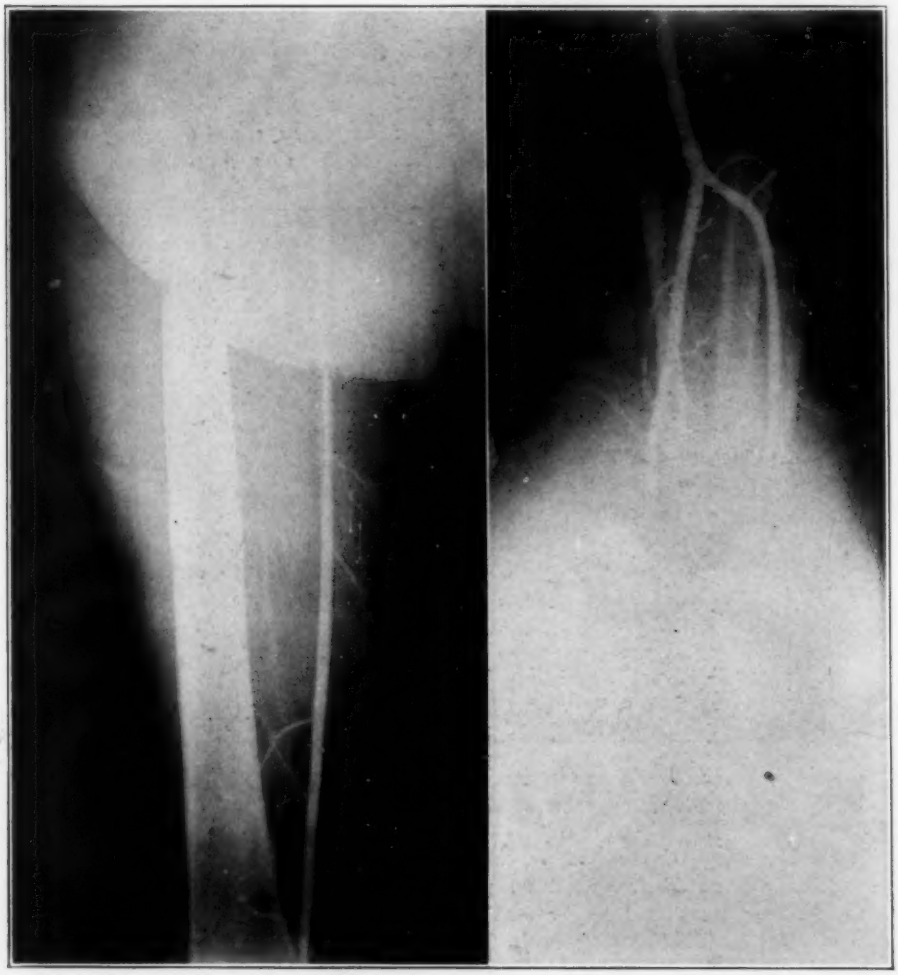

Fig. 4 (case 4).—Arterial tree showing case of moist gangrene spreading higher following arterial injection.

feeling of numbness in both feet and inability to move them as he had formerly done. The right foot showed a small gangrenous mass on the first and second toes. The great toe was involved to the size of about a five cent piece. Anesthesia involved all the toes of the foot. Both feet were numb, and the patient was unable to flex the metarsal and phalangeal joints. There was

some pain involving the affected area. Touch and pressure sense was lost over the discolored portions, but pain, especially on the application of heat, was present. This numbness of the toes had become progressively worse during the last several years. The numbness during the last few days began to involve the right hand and fingers, particularly the fifth finger. The predominant loss was that of the sense of touch. The condition in the left leg became

Fig. 5 (case 5).—Arterial tree in case of senile gangrene. One view shows the leg upside down. While the main arterioles are open in both anterior and posterior tibias, senile gangrene occurred in the foot due to the obstruction of the arterioles and capillaries.

progressively worse while the patient was in the hospital. It became cold, motion was lost, and the leg was painful. On February 8, 15 cc. of 100 per cent sodium iodine was injected into the left femoral artery, and roentgeno-

grams made of the leg and foot after injection of sodium iodide into the femoral artery indicated a good arterial supply to the leg. The medium stayed in the arteries for a considerable length of time, none of it being in the arteries after an hour, however, except probably a small amount which stuck to the wall. On February 12, the skin showed a mottling appearance up to the knee and this mottling was extending medially to the perineum. On February 17, the leg had increased markedly in size and had apparently lost all sensation. A definite line of demarcation was not present. The odor was typically

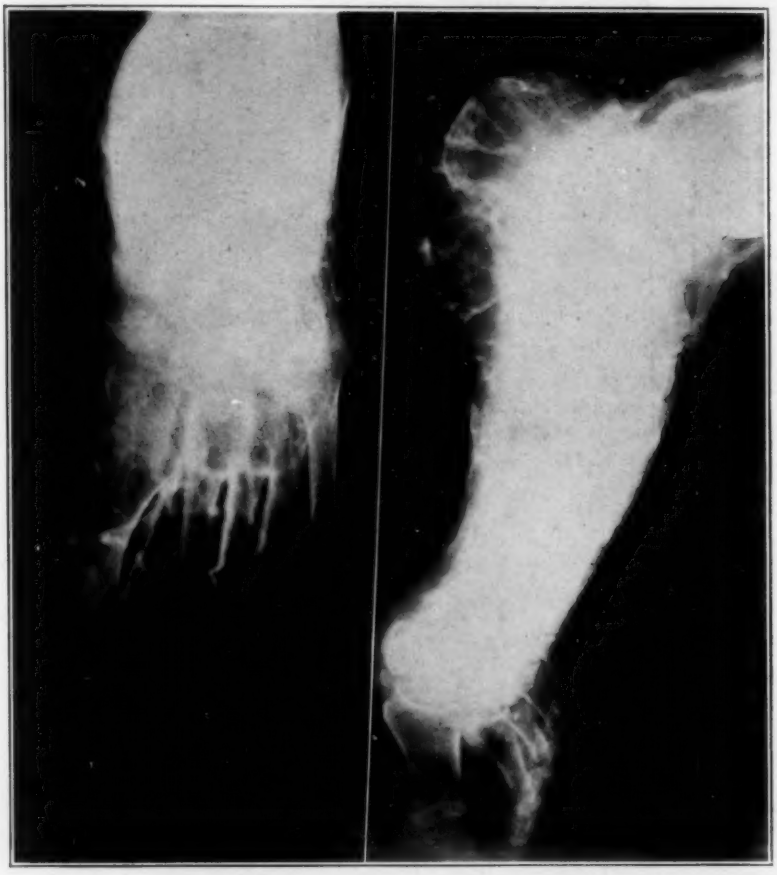

Fig. 6 (case 6).—Arterial tree in the foot showing arterioles blocked by disease in a case of senile gangrene. The solution is seen being retained in the arterioles and capillaries.

gangrenous. On February 20, the lower half of the thigh in front and the thigh to the buttocks posteriorly showed moist gangrene. The patient was unconscious. The blood pressure was decreased, and catherization was necessary. The patient recovered ability to void, but showed no particular improvement. He died on March 1.

In this case also the results were unfavorable, and it was again demonstrated that sodium iodide is dangerous if there is sufficient obstruction to cause its

retention in the vessels for an unduly long time. In arteriosclerotic gangrene, as previously mentioned, the capillaries and arterioles are markedly narrowed, and unquestionably in this type of case, the injection is not to be used.

SUMMARY

1. Roentgenologic examination after intra-arterial injection of sodium iodide accurately reveals the condition of the blood vessels. Such injection is apparently harmless when there is good capillary circulation and a free return of blood into the general circulation. The method is apparently safe.

2. In aneurysms of the extremities, both arterial and arteriovenous, valuable information is gained in regard to the blood vessels entering and leaving the aneurysms and the collateral circulation, which may be of great assistance in determining the method of treatment.

3. In spontaneous gangrene resulting from venous or arterial obstruction, the method is not safe and probably should not be used. It is contraindicated in moist gangrene with venous obstruction, and also in arteriosclerosis because of the narrowing in the arterioles and capillaries. In thrombo-angiitis obliterans, the capillaries are usually much more normal, and there is better collateral circulation; therefore, in this condition, the method should not be harmful.

STUDIES IN INTESTINAL OBSTRUCTION

III. SIMPLE OBSTRUCTION: A STUDY OF THE CAUSE OF DEATH IN MECHANICAL OBSTRUCTION OF THE UPPER PART OF THE INTESTINE *

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There are two fairly distinct types of intestinal obstruction, clinically as well as experimentally; in one the continuity of the intestinal canal has been interrupted; in the other gross evidence of damage to the obstructed segment exists in addition to a break in the continuity of the tract. These forms have aptly been described as simple and strangulation obstruction.[1]

Early clinicians and investigators thought that in simple occlusion of the bowel, the wall became permeable to bacteria in the intestine, and peritonitis and bacterial invasion of the organism brought about a lethal outcome.[2] Such a contention has been set aside by subsequent investigation, as well as by the frequent opening of the abdomen, under aseptic conditions of both patients and dogs with obstruction.

Amussat [3] is generally looked on as being the originator of the intoxication theory for the explanation of symptoms and death in intestinal obstruction. This view, which today has most investigators and clinicians as its adherents, accounts for the lethal outcome in the formation and absorption of potent toxins above the point of obstruction. Braun and Borrutau,[4] unable to verify the presence of a toxic body in the obstructed intestine that is not present normally, ascribed death to splanchnic paresis and shock. Hartwell and Hoguet [5] inclined

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* From the Department of Surgery of the University of Minnesota.

* Presented before the Minnesota Pathological Society.

1. Hausler and Foster: Studies on Acute Intestinal Obstruction; II. Acute Strangulation, Arch. Inter. Med. **34**:697 (Nov.) 1924.

2. Khautz, A.: Zur Frage der Bakteriamie bei Ileus und postoperativen Darmlähmung, Arch. f. klin. Chir. **88**:412, 1909. Börzeky, C., and Generisch, A.: Beitrage zur lokal diagnose der inneren Darm Okklusionen und zur Frage der Autointoxikation, V, Bruns Beitr. z. klin. Chir. **36**:448, 1902.

3. Amussat: Relation de la maladie de bouroussais, Paris, 1838, quoted by Enderlen and Hotz: Ueber die Resorption bei Ileus und Peritonitis, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **23**:755, 1911.

4. Braun, W., and Borrutau, H.: Experimental kritische Untersuchungen über den Ileus Tod, Deutsche Ztschr. f. chir. **96**:544, 1908.

5. Hartwell, J. A., and Hoguet, J. P.: Experimental Intestinal Obstruction in Dogs with Especial Reference to the Cause of Death and the Treatment by Large Amounts of Normal Saline Solution, J. A. M. A. **59**:82 (July 13) 1912.

to the belief that dehydration in itself is the most important factor. Hausler and Foster [6] stated that death in cases of simple occlusion of the bowel can be accounted for on the basis of starvation alone.

Since Hartwell and Hoguet [5] first made known the efficacy of saline in prolonging the life of the animal in which an obstruction had been established experimentally, this observation has been corroborated by a large number of investigators and clinicians.[7] Extensive experimentation by Haden and Orr relative to the protection that sodium chloride solution afforded the animal [8] with intestinal obstruction would indicate that the virtue of the saline lay in the drug per se. They were unable to obtain the same beneficial effect from other drugs containing either the sodium or the chloride radical alone. They believed that sodium chloride combines with the toxic bodies responsible for the fatal issue in intestinal obstruction, rendering them inert.

In a previous study,[9] by experiments on animals we were unable to ascertain the presence of toxic bodies in the intestine with simple obstruction not found in the normal intestine. The intestinal contents of dogs with an obstructed intestine to which salt solution had been administered were also toxic on injection. This would indicate that unusual toxins, if present, were not rendered innocuous within the bowel.

In another study [10] it was found that the intestine, with interruption of its continuity, was not abnormally permeable to histamine. In this investigation we have been concerned only with simple obstruction with particular reference to the evidence that abnormal absorption from the obstructed bowel is responsible for the death of such an animal.

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6. Hausler and Foster: Studies on Acute Intestinal Obstruction: III. Simple Obstruction, Arch. Inter. Med. **36**:31 (July) 1925.

7. Dixon, C.: The Value of Sodium Chloride in the Treatment of Duodenal Intoxication, J. A. M. A. **82**:1498 (May 10) 1924. Haden and Orr: Chemical Changes in the Blood of Man After Acute Intestinal Obstruction, Surg. Gynec. & Obst. **37**:465, 1923; Hausler and Foster (footnote 6). MacCallum, W. B.; Lintz, H.; Vermilye, H. N.; Legett, T. H., and Boas, E.: The Effect of Pyloric Obstruction in Relation to Gastric Tetany, Bull. Johns Hopkins Hosp. **31**:1, 1920. McVicar, C. S.: The Clinical and Laboratory Findings in Certain Cases of Obstruction on the Upper Gastro-Intestinal Tract: The Rôle of Blood Chemistry in Diagnosis, Prognosis and Treatment of this Condition, Am. J. M. Sc. **169**:224, 1925.

8. Haden, R. L., and Orr, T. G.: Chemical Changes in the Blood of the Dog After Obstruction on the Duodenum, J. Exper. Med. **37**:365 and 377, 1923; ibid. **39**:321, 1924; ibid. **38**:55, 1923.

9. Wangensteen, O. H., and Chunn, S. S.: Studies in Intestinal Obstruction. A Comparison of the Toxicity of Normal and Obstructed Intestinal Content, Arch. Surg., to be published.

10. Wangensteen, O. H., and Loucks, M.: Studies in Intestinal Obstruction; II. The Absorption of Histamine from the Obstructed Bowel, Arch. Surg., to be published.

As considerable experimentation has been done in this field, a number of the experiments reported in this study have been performed by other investigators. In order to be able to interpret the results of such experi-

Data on Dogs in Experimental Intestinal Obstruction

Dog	Nature of Procedure	Day After Operation	Blood Mg. per 100 Cc.		Urine and Vomitus	
			Urea	Chlorides	Amount	Nonprotein Nitrogen
82	Duodenal obstruction of the severed	0	11.80	540	125	0.90
Weight,	intestine, 12/22/26	1	26.60	450	1,200	8.57
40 pounds		2	19.20	430	315	4.41
(18.1 Kg.)		3	40.13	430	550	5.66
		4	38.73	360	160	2.56
		5	41.82	340	500	5.32
		6	59.20	310	520	5.04
		7	83.16	320	540	4.10
Died 12/29/27; weight at death, 32 pounds (14.5 Kg.)						
83	Duodenal obstruction of the severed	0	10.26	490	90	1.10
Weight,	intestine, 12/22/26; given 400 cc. 2%	1	19.13	490	365	4.96
29 pounds	saline subcutaneously once daily	2	21.46	450	1,500	6.44
(13.2 Kg.)		3	18.66	560	1,010	7.98
		4	15.86	740	2,200	6.57
		5	16.80	560	2,225	7.52
	Salt discontinued	6	20.53	520	2,700	5.22
		7	15.86	590	870 *	6.08
		8	27.30	430	1,850	5.58
		9	18.26	410	1,025	4.37
		10	17.87	390	1,300	3.95
		11	14	470	1,525	6.40
		12	16.30	520	740	3.33
		13	14.47	410	110	1.54
		14	16.54	400	160	3.20
		15	15.38	...	265	2.30
		16	18.20	310	120	2.40
		17	16.40	310	60	1.20
		18	.....	...	100	2.40
Dog died 1/10/27						
94	Duodenal obstruction of the severed	0	14	530	.....	....
Weight,	intestine and gastroenterostomy,	1	14.47	400	2,800	4.42
39 pounds	1/6/27; no saline given	2	28.36	380	2,000	3.38
(17.7 Kg.)		3	25.20	250	2,000	5.27
		4	18.20	310	440	4.52
		5	56.93	260	1,000	6.30
		6	84.46	260	450	4.95
		7	119.06	250	870	7.40
		8	.....	...	1,750	6.70
Died; weight, 29 pounds						
116	Duodenal obstruction and gastro-	0	9	500	125	1.20
Weight,	enterostomy, 2/7/27; saline given	1	14	440	450	5.37
40 pounds	subcutaneously, 400 cc. 2% once	2	16.08	430	2,700	6.60
	daily	3	15.78	360	8,200	8.76
		4	14.32	510	1,575	3.70
		5	17.75	250	1,400	4.56
	Salt discontinued	6	9	430	650	3.40
		7	18.60	410	500	3.87
		8	16.50	440	50	1.25
		9	15.86	370	560	2.43
		10	17.40	430	600	2.60
		11	23.20	460	300	1.96
		12	14	420	250	1.75
7/7/27, dog in satisfactory condition; weight 34 pounds (15.4 Kg.); vomits occasionally						

* In these experiments the urine and vomitus were collected together. The dogs were allowed to drink water.

ments with any judgment, we felt that it was necessary to make our own observations. Since the results of a number of the experiments to be cited are fairly well known and established, the experimental procedures will not be listed in detail.

METHOD

All experiments were performed on dogs under ether anesthesia and aseptic technic. Severed gut obstructions were established largely in the duodenum; a few were established in the lower ileum and one in the lower part of the descending colon. At the point selected for obstruction, the gut was severed between crushing clamps [11] and the ends of the bowel inverted [12] much as the stump of the appendix is treated in appendectomy.

The effect of giving salt solution was noted in some of the dogs with duodenal obstruction. In several animals gastro-enterostomy was established in addition to the high duodenal obstruction. Some of these animals were given salt solution; others were not. Division of the stomach beneath the diaphragm was practiced in a few animals with and without the subcutaneous administration of salt solution. In two animals the esophagus was ligatured in the neck. Duodenal fistulas were established in a number of animals. Salt solution was given to some of them.

When sodium chloride was administered, it was given subcutaneously, with a 50 cc. syringe. In most instances, from 250 to 400 cc. of a 2 per cent solution was given once a day. A 1 per cent solution was given to a few animals, 400 cc. being given twice a day. The blood urea and chlorides were determined daily on all animals. Blood was obtained from the jugular vein or the vein on the outer aspect of the hind leg of the dog. Some of the animals were placed in metabolism cages and the nitrogen output in the urine determined. The nonprotein nitrogen in the urine was determined by the micro-Kjeldahl method of Folin and Denis [13] and the urea nitrogen of the blood by the method of Van Slyke and Cullen.[14] The blood chlorides were determined according to the method described by Gettler.[15]

Two of three dogs in which severed gut obstructions were established in the duodenum about 15 cm. beyond the pylorus died within seventy-two hours. The increase of the nonprotein nitrogen in the blood and decrease of the chlorides observed by previous investigators [16] were

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11. In some earlier experiments, it had been observed that animals occasionally survived ligature obstructions. In the animals that died or were killed, the obstruction was usually found incomplete at necropsy.

12. The distal end of the bowel, when the division was made in the small intestine, was anchored to the abdominal wall because it was noted that in some of the early animals in which this procedure was not practiced, intussusception of the distal loop not infrequently occurred.

13. Folin, O., and Denis, W.: Nitrogen Determination by Direct Nesslerization; I. Total Nitrogen in Urine, J. Biol. Chem. **25**:473, 1916.

14. Van Slyke, D. D., and Cullen, G. E.: A Permanent Preparation of Urease and Its Use for Rapid and Accurate Determination of Urea, J. A. M. A. **62**:1558 (May 16) 1914.

15. Gettler, A. O.: A Method for the Determination of Death by Drowning, J. A. M. A. **77**:1650 (Nov. 19) 1921.

16. Cooke, J. V.: Rodenbaugh, F. H., and Whipple, G. H.: Intestinal Obstruction; A Study of Non-Coagulable Nitrogen of the Blood, J. Exper. Med. **23**:717, 1916. Connors, J. F.; Killian, J. A., and Eisberg, H. B.: Chemical Changes in the Blood in Intestinal Obstruction, Proc. Soc. Exper. Biol. & Med. **20**:357, 1923. Haden and Orr (footnote 8, first reference). Tillotson, W., and Comfort, C. W.: The Total Nonprotein Nitrogen and the Urea of the Blood in Health and in Disease as Estimated by Folin's Methods. Arch. Inter. Med. **14**:620 (Nov.) 1914.

noted. The third dog survived the procedure seven days.[17] Two dogs with obstruction in the lower part of the ileum survived four days. In a group of ten dogs given from 250 to 400 cc. of a 2 per cent salt solution subcutaneously, the average length of life following duodenal severed gut obstruction was seven days. Two animals with this type of obstruction, were given saline solution for one week; one animal survived eighteen days, the other, nineteen.

Severed obstructions of the same nature were made in two dogs, and a posterior gastro-enterostomy was added beyond the duodenojejunal flexure. One of these animals survived two days; the other, nine. The changes in the blood chemistry present in animals with simple duodenal occlusion were observed. In two other animals, severed duodenal obstructions were established, gastro-enterostomy was performed and a linear division of the pyloric sphincter muscle (Rammstedt procedure) without incising the mucosa was practiced. The outcome was the same as in the animals in which the pyloric sphincter was intact; both died within seventy-two hours. Three dogs with a severed duodenal obstruction and an added gastro-enterostomy, were given saline subcutaneously for a week. One died two weeks after the original procedure from an obstruction due to an adhesive band lower in the intestinal tract. Another died from an extensive slough over the hind quarters where the hypertonic saline had been injected twelve days after the obstruction was established. The other dog was alive and well almost six months later. This dog became somewhat emaciated during his convalescence and vomited considerably. Occasional vomiting was noted at the last observation. Material alteration in the blood metabolites was not observed in any of the latter animals to which salt solution was given temporarily. An increased excretion of nitrogen, however, was observed in the urine.

Complete duodenal fistulas were established in three animals at the same level at which obstruction had been established in the other dog. The proximal segment was drawn out through a small stab wound and sutured to the abdominal wall, leaving the lumen patent. The distal end was inverted and anchored to the parietal peritoneum. These dogs died three days later. The changes in the blood chemistry noted by Walters, Kilgore and Bollman,[18] were observed in these animals. Two of three other animals in which the same operative procedure was performed but which were given saline solution survived eight days, the other lived five days.

By making a T incision in the left rectus muscle in one dog, easy access was had to the cardiac end of the stomach. This was divided as

17. Throughout this work on intestinal obstruction, it has been noted that larger dogs stand obstruction better than small animals.

18. Walters, W.; Kilgore, A. M., and Bollman, J. L.: Changes in the Blood Resulting from Duodenal Fistula, J. A. M. A. **86**:186 (Jan. 16) 1926.

high as possible and both proximal and distal ends inverted. The dog lived one day. In another animal the same procedure was repeated, but 400 cc. of water was poured into the peritoneal cavity. The outcome was the same. In four other animals 400 cc. of a 2 per cent sodium chloride solution was given once a day following the same procedure. One of these animals survived for a day; another, two days; a third dog lived seven days, and the other twelve days. Two dogs with ligation of the cervical esophagus died within two days.

In one animal, the descending colon was divided as low down as possible and the ends turned in. This dog survived the procedure for forty-two days. The increased nonprotein nitrogen found in the blood of animals with severed duodenal obstruction was not observed in this animal. The nitrogen excretion in the urine, however, was increased. At death the animal was markedly emaciated. The entire intestine was enormously distended, and the lower portion of the obstructed colon was packed tight with fecal material.

Two dogs were starved and dehydrated by being deprived of water for three days. Severed gut duodenal obstructions were then established. One of these animals survived for eight days. The changes in the blood chemistry found in dogs with simple duodenal obstruction were also observed in these animals. Two dogs with ileal obstructions were similarly starved and dehydrated without materially prolonging their lives.

Two dogs were dehydrated by being deprived of water and were given subcutaneous injections of pilocarpine and apomorphine to induce vomiting and added dehydration. Low values for blood chlorides were obtained in these animals without elevation of the blood urea nitrogen.

The nitrogen output of the animals kept in metabolism cages was consistently high following duodenal obstructions. The urinary excretion of nitrogen was also increased in the animals that were given salt solution subcutaneously, and in which no material increase of the blood nonprotein nitrogen was observed.

COMMENT

Clinicians and investigators alike concede the more serious import of obstructions high in the intestinal tract. In these experiments it has been recounted that dogs with duodenal obstruction usually die in seventy-two hours, whereas a dog with a severed gut obstruction low in the colon survived the obstruction for forty-two days. Starvation could well be assigned as the cause of death of the latter animal; in the case of the former, it is generally held that the formation and absorption of a potent toxin is responsible.

When, however, saline is administered to the animal with duodenal obstruction, his life may be prolonged for three weeks, as many investi-

gators have observed; if, however, saline is given for only a week, the dog lives just as long. Two dogs with several duodenal obstructions were given salt solution for only six days; following the establishment of the obstruction, death was deferred eighteen days in one and nineteen days in the other. Hausler and Foster [6] have made the same observation. By discontinuing the administration of saline after four or five days, two of their animals lived twenty-one and twenty-eight days, respectively, after duodenal obstructions had been established.

If the saline acts so as to detoxify an absorbed poisonous substance in the animal with obstruction high up in the intestine, why does he not die promptly following the discontinuance of the saline therapy? Certainly the temporary administration of salt solution has not permanently diminished the permeability of the intestine to toxic substance. It is apparent, therefore, that the formation and absorption of potent toxins alone cannot account for the difference in behavior of the animal with low and high obstruction.

Kader [19] has shown that when the animal dies in a few days, there is no great distention of the abdomen or of the entire intestinal tract (dogs, cats, pigs, and horses) with simple obstructions in the small intestine. The abdomen usually measures only 1 inch more in diameter than when the obstruction was established. Only the segment of intestine immediately above the obstruction shows much dilatation. In the patient or animal with obstruction in the lower part of the colon a progressive and marked distention of the entire intestinal tract gradually takes place. When this dilatation, in which the entire intestine eventually participates, has occurred, there must exist the same possibility for the formation of toxins high in the intestine that exists when the mechanical hindrance is located at a higher level. At the same time the factor of absorption should be tremendously increased over that present when a much shorter segment is obstructed. An animal with such an obstruction can live a long time, and when death does occur, the chemical changes present in the blood of animals with high intestinal obstruction are absent.

Braun and Borrutau [4] found that dogs with complete duodenal fistulas did not live any longer than dogs with intestinal obstruction at the same level. Walters, Kilgore and Bollman [18] have shown that the same chemical changes occur in the blood of animals with duodenal fistulas as in the blood of animals with duodenal obstruction. Walters and Bollman [20] have observed the same in animals with gastric fistulas. In such an experiment, the factor of absorption is done away with

19. Kader, B.: Ein experimenteller Beitrag zur Frage des localen meteoresmus bei Darm Occlusion, Deutsche Ztschr. f. Chir. **33**:57, 1892.

20. Walters, W., and Bollman, J. L.: Experimental Acute Gastric Fistula, Arch. Surg. **13**:578 (Oct.) 1926.

entirely, but the animal dies from the loss of stomach and duodenal contents. Murlin and Sweet [21] stated that Pavlow had observed that dogs with complete pancreatic fistulas die, and that Pavlow could keep them alive only by the administration of sodium. That experimental complete external pancreatic fistulas may prove fatal, has recently been observed again by Elman and McCaughan.[22]

Haden and Orr [23] have found that dogs with esophageal obstruction die even more quickly than animals in which the duodenum is obstructed. The same increase in the nonprotein blood nitrogen and fall in chlorides were observed that occur in duodenal obstruction. A marked increase in the excretion of nitrogen in the urine was also present.[24]

We have found that subcutaneous administration of saline may also prolong the life of the animal with obstruction at the cardiac end of the stomach and prevent the marked increase of nonprotein nitrogen in the blood. We were able to prolong the life of one such animal for twelve days. When the esophagus was obstructed in the neck, early death resulted. In these animals, the loss of gastric contents by vomiting is at once ruled out. At the same time, the factor of absorption is reduced to a minimum. Walters and Bollman [19] have noted the high chloride value of the content of the bowel in a dog with excision of the stomach and inversion of the esophageal and duodenal stumps.

Over a period of four years, during which time one of us (O. H. W.) has been on the lookout for these blood changes in patients with esophageal obstruction, two such cases have been observed. One patient had obstruction in the upper part of the esophagus, the other in the lower part of the esophagus; both showed markedly increased blood urea nitrogen, without clinical evidence of renal insufficiency. Dehydration and starvation were occurring at a rapid rate.

In another patient with marked vomiting following gastro-enterostomy, in whom changes in the blood chemistry were present which accompany pyloric obstruction, it was observed that on two consecutive days the vomitus and retention of fluid aspirated from the stomach exceeded by far the total intake of fluid. This patient was not allowed water by mouth. Fluid was given to the extent of 4,000 cc. of physiologic sodium chloride solution by hypodermoclysis on two succes-

21. Murlin, J. R., and Sweet, J. L.: Pancreatic Diabetes in the Dog; the influence of Pylorus Exclusion and of Gastrectomy Upon the Effects of Pancreatectomy, J. Biol. Chem. **28**:261, 1916.

22. Elman, R., and McCaughan, J. M.: On the Collection of the Entire External Secretion of the Pancreas Under Sterile Conditions and the Fatal Effects of Loss of Pancreatic Juice, J. Exper. Med. **45**:561, 1927.

23. Haden and Orr: Chemical Changes in the Blood of the Dog After Obstruction of the Cardiac End of the Stomach, J. Exper. Med. **38**:477, 1923.

24. Haden and Orr: The Excretion of Nitrogen After Upper Gastro-Intestinal Tract Obstruction, J. Exper. Med. **45**:433, 1927.

sive days. The urinary output was also low, and almost 6,000 cc. was returned by vomiting or by aspiration of the stomach with a tube on each of these two days. Unfortunately, an analysis for chlorides was not made on the fluid. Following the subcutaneous administration of saline the changes observed in the blood chemistry returned to their usual values, and the patient gradually ceased vomiting and recovered.

Undoubtedly both chlorides and fluid may be lost into the bowel when an obstruction exists high in the intestinal tract. Dr. George Fahr [25] had the intestinal content in a patient with acute intestinal obstruction of the mechanical type drained off through an enterotomy opening and examined for salt content. Analysis showed sodium chloride present in a 10 per cent concentration. Snapper [26] has suggested that the chloride may be lost in the body tissues in pneumonia when the blood chlorides are low. He postulated a disturbance between the interchange of salt between tissue and body fluids such that the permeability for the chloride ion is increased. The low blood chlorides in obstruction could be partially accounted for in such an explanation.

It has been demonstrated that in experimental dehydration of animals, a moderate increase in the nonprotein nitrogen of the blood and an increase in nitrogen excretion can be brought about.[27] Lois and Eaton Mackay [28] obtained marked increases in the nonprotein nitrogen of rabbits dehydrated by intravenous injections of sucrose. In the dehydrations occurring normally in some infants a marked increase in nonprotein blood nitrogen has been noted.[29] In the animals that we caused to be dehydrated and to vomit by the administration of pilocarpine and apomorphine, the extreme increases in the nonprotein nitrogen of the blood observed in experimental obstructions were not obtained. The rapidity of dehydration and loss of salt must be greater in an animal in which a constant obstruction is present than in an animal deprived of water and given apomorphine and pilocarpine four times in ten hours and then left alone for fourteen hours.

Gastro-enterostomy performed on the dog with severed duodenal obstruction does not prevent the animal's death or obviate the chemical changes in the blood. Linear division of the pylorus at the same time

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25. Fahr, George: Personal communication.

26. Snapper, J.: Ueber den Zusammenhang zwischen Funktion der Nieren und Chlorretention bei fieberhaften Krankheiten, Deutsches Arch. f. klin. Med. **111**:430, 1913; Ueber eine permeabilitäts Anderung der Zellen als Ursache der Chlorretention bei fieberhaften Krankheiten, ibid. **111**:441, 913.

27. Keith, N. M.: Experimental Dehydration, Am. J. Phys. **68**:80, 1924.

28. Mackay, Lois, and Mackay, Eaton, M.: Increased Blood Urea Concentration of Extrarenal Origin, Am. J. Phys. **70**:394, 1924.

29. Bessau, G.; Rosenbaum, S., and Leichentritt, B.: Beiträge zur Säuglings-Intoxikation, Monatschr. f. Unfalheilk. **23**:46, 1922.

does not affect the outcome. When, however, sodium chloride solution is given to such an animal for a short period, changes in the blood do not occur, and he is able to live for a long time. Such an animal continues in good health except for the emaciation incident to the vomiting occasioned by the obstructed segment.

The virtue of sodium chloride in the treatment of the animal with a fistula or obstruction high in the intestinal tract probably represents, therefore, replacement therapy. A protective mechanism exercised through a detoxifying agency is unlikely. McCallum and his co-workers [30] and Gamble and Ross [31] are also of this opinion. It has been demonstrated by Haden and Orr [32] that other combinations of the sodium or chloride radical will not ward off early death in animals with duodenal obstruction. Hypertonic dextrose solutions similarly fail to do so. Walters and Bollman [19] have made the same observation in dogs with duodenal and gastric fistulas. Just why sodium chloride apparently alone should be able to fulfil this function is difficult to surmise, but the suggestion of Gamble and Ross [31] that it is particularly suited to replace the lost fluid and fixed base is probably correct.

Whipple and his co-workers [33] have shown that an increased destruction of protein occurs in intestinal obstruction. Haden and Orr [23] have made the same observation. Though injury to the kidney obtains in simple occlusion of the bowel, it is not likely that the increase in nonprotein nitrogen of the blood is due to renal insufficiency, as has been suggested.[34] The fact that nitrogen is excreted in excess and that phenolsulphonphthalein is normally excreted [35] shows that renal failure is not present.

The increased excretion of nitrogen probably partakes of an acceleration of general body breakdown of protein. At any rate, the necrosis of the ends of the inverted bowel is not in itself responsible for the increased destruction.

An experiment often cited to indicate that death in obstruction is due to the absorption of potent toxins from the intestine is that per-

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30. MacCallum, W. B.; Lintz, H.: Vermilye, H. M.; Legett, T. H., and Boas, E. (footnote 7, third reference).

31. Gamble, J. L., and Ross, S. G.: The Factors in the Dehydration Following Pyloric Obstruction, J. Clin. Investigation **1**:403, 1925.

32. Haden and Orr (footnote 8, first and second reference).

33. Whipple, G. H.; Cooke, J. V., and Stearns, T.: Proteose Intoxications and Injury of Body Protein, J. Exper. Med. **25**:479, 1912.

34. Brown, G. E.; Eusterman, G. B.; Hartman, H. R., and Rowntree, L. G.: Toxic Nephritis in Pyloris and Duodenal Obstruction; Renal Insufficiency Complicating Gastric Tetany, Arch. Inter. Med. **32**:425 (Sept.) 1923.

35. Connors, J. F.; Killian, J. A., and Eisberg, M. B. (footnote 16, second reference). Louria, H. W.: The Blood Urea Nitrogen in Acute Intestinal Obstruction, Arch. Inter. Med. **27**:620 (May) 1921.

formed by Sauerbruch and Hyde.[36] They denuded the abdominal wall of two rabbits and sutured the skin of one to that of the other. Later, when a good crossed circulation had been established, an obstruction was created in one, following which the same toxic symptoms and death also occurred in the animal in which obstruction had not been established. The outcome undoubtedly would have been the same if a duodenal fistula had been established in one of the animals.

Sugito [37] claims to have isolated a toxin in the blood returning to the liver from the intestine in the mesenteric veins in dogs with intestinal obstruction. When the serum from blood thus obtained was injected into the peritoneal cavity of rats, toxic symptoms were observed that were not elicited following the injection of blood from animals without obstructions. Wilkie [38] and McLean and Andries,[39] however, have transfused large amounts of blood from dogs with intestinal obstruction to normal animals, without any untoward effect. Wilkie also removed the intestinal contents from a dog with obstruction and placed them in the obstructed intestine of another dog without hurrying the development of toxic symptoms.[40] A number of investigators [41] have demonstrated that intestinal contents obtained from animals with intestinal obstruction do not elicit toxic symptoms when placed in the bowel of a normal animal.

Schönbauer [42] and Chenut [43] contended that abnormal absorption in simple obstruction may occur via the peritoneal cavity. Schönbauer stated in support of his contention that iodine placed in the bowel of a dog with intestinal obstruction can be recovered from the peritoneal cavity. He believed that death during obstruction is due to toxins permeating the wall of the bowel and being absorbed from the peritoneal cavity rather than to abnormal absorption from the lumen of the intestine. Schönbauer and Löffler stated [44] that the antitryptic titer of the blood of

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36. Sauerbruch, F., and Hyde, M.: Weitere Mitteilingen ueber die parahiose bei Warmbeitern mit versuchen ueber Ileus and Urämie, Ztschr. f. Exper. path. u. Therap. **6**:33, 1906.

37. Sugito, S.: Ueber die Todesursache bei Ileus (Intoxicationstheorie), Mitt. a. d. med. Fak. d. k. Univ. Kyushu. u. Fukuoka **9**:229, 1924.

38. Wilkie, D. P. D.: Acute Intestinal Obstruction, Lancet **1**:1135, 1922.

39. McLean, A., and Andries, R. C.: Ileus Considered Experimentally, J. A. M. A. **59**:1614 (Nov. 2) 1912.

40. Wilkie, D. P. D.: Experimental Observations on the Cause of Death in Acute Intestinal Obstruction, Brit. M. J. **2**:1064, 1913 (footnote 38).

41. Chenut, A.: L'Eperimentation dans l'occlusion mechanique du jejunoileon, Rev. d. chir. **45**:474, 1926 (footnote 38).

42. Schönbauer, L.: Die Fermente in Ihrer Beziehung zu gewissen der Gallenblase und zum Ileus, Arch. f. klin. Chir. **130**:427, 1924.

43. Chenut (footnote 41, first reference).

44. Schönbauer, L., and Eöffler, E.: Ueber Ileus-serum, Experimentelle und Klinische Untersuchungen, Wein. klin. Wchnschr. **38**:135, 1925.

patients suffering from intestinal obstruction is increased, and they recommended the use of an antitryptic ferment. Chenut [43] has examined histologically the intestinal wall in cases of simple obstruction and has observed evidence of damage to the mucosa at and above the level of obstruction. He found muscular and mucous and submucous layers thinned out. The epithelium over the intestinal villi appeared desquamated in areas.

In strangulating types of obstruction when gross evidence of damage to the intestinal wall such that the bowel is no longer viable is present, the bowel undoubtedly becomes permeable to the products of autolysis of the wall of the bowel consequent on the strangulation and to the contents within the lumen as well. When the viability of the wall of the bowel in simple obstruction is threatened by the distention of the intestine, permeation of the wall by toxins and absorption via the peritoneal cavity no doubt may also occur. As has been pointed out previously,[10] however, it is unusual to observe destructive changes in the wall of the bowel during simple occlusion. Distention ulcers or the gangrenous patches that occur on the antimesenteric border of the strangulated intestine are infrequently observed in simple obstruction. We have observed them frequently in closed loop obstruction with the continuity of the remainder of the tract reestablished. The tension in such a loop with both ends closed must, therefore, be much greater than in simple severance of the continuity of the bowel.

The fact that dogs with an obstruction high up in the severed gut can live for three weeks after having been given saline solution subcutaneously for a few days militates against the theory of the migration of toxins through the wall of the bowel and their absorption via the peritoneal cavity in simple obstruction. A great amount of toxin was not absorbed through the distended wall of the bowel in the animal with an enormously distended intestine which lived forty-two days following severed gut obstruction in the descending colon. The absorption from the bowel via the peritoneal cavity could also not have been great in the animals with duodenal occlusion which survived the procedure for almost three weeks after a few days of subcutaneous administration of salt solution and in which the obstructed segments were found uniformly markedly dilated.

A denial is not made of the suggestion that in simple intestinal obstruction absorption may be increased over that present in the normal bowel; but evidence of it has not been elicited in these experiments. The more rapid death when the obstruction occurs high up in the intestine, however, is not accounted for by the formation and absorption of a more potent toxin than that present when the obstruction is low down in the bowel; the more likely cause is the rapid dehydration and loss of body chlorides attending obstruction of the upper part of the intestinal tract.

That liver insufficiency does not play a great rôle in simple obstruction, as has been suggested,[45] is apparent from the injections of histamine into the mesenteric veins of dogs with intestinal obstruction, described in a previous paper. The liver of animals with intestinal obstruction still possessed the same slightly detoxifying action for histamine observed in the liver of normal animals. The presence of a high blood fibrin in animals with an obstruction in the upper part of the intestine, as observed by Haden and Orr,[46] is also against such an assumption.

The increase in the excretion of nitrogen in the urine could be due to a toxic action occasioned by abnormal absorption; but the same obtains in obstruction of the cervical esophagus. Is it likely that absorption plays any great rôle in the death of such an animal?

SUMMARY

Obstruction of the upper part of the intestine is much more serious than obstruction low down in the colon. An animal with the former type of obstruction dies sooner because dehydration and loss of chlorides occur more rapidly. Subcutaneous administration of saline to such an animal prolongs his life and obviates the alteration in blood chemistry noted in animals with duodenal obstructions to which salt solution is not given. An increased excretion of nitrogen, however, also occurs in the urine of animals with duodenal obstruction to which salt has been given. A temporary administration of saline is just as efficient in prolonging the animal's life as continued daily administration. Therefore, the virtue of the remedy cannot lie in any detoxifying mechanism. The fact that animals with gastric or duodenal fistulas die as quickly as dogs with obstructions at the same level and with the same changes in the chemistry of the blood and increase in nitrogen excretion in the urine, and the fact that the administration of saline prolongs the life of such an animal also indicate that the virtue of the drug lies in substitution therapy. Dogs with duodenal obstruction on which gastro-enterostomy has been performed can live indefinitely when given salt solution for a few days. In such an animal, the conditions are right for permanent recovery when the dehydration and the loss of the contents of the stomach incident to the obstruction have been ameliorated by the administration of saline. The animal with esophageal obstruction does not lose chlorides and fluid by vomiting, but the life of the animal can be prolonged by the administration of saline. On the same basis of loss of fluid and fixed base from the body, some alteration in the permeability of tissue is necessary to

45. Werelius, A.: Is Death in High Intestinal Obstruction Due to Liver Insufficiency? J. A. M. A. **79**:535 (Aug. 12) 1922.

46. Haden and Orr: Blood Fibrin in Upper Gastro-Intestinal Tract Obstruction, J. Exper. Med. **45**:427, 1927.

explain the death of an animal with esophageal obstruction or a rabbit with duodenal obstruction that does not vomit. In the animal with ileal or colonic obstruction the marked increase of nonprotein blood nitrogen and low values for blood chlorides are not obtained. The administration of saline in low obstruction would therefore not afford the same protection as it does to animals with duodenal obstruction.

CONCLUSIONS

1. The explanation of the rapidly fatal issue in dogs with obstruction in the upper part of the intestine is to be found in the rapid dehydration and loss of chlorides accompanying occlusion of this portion of the bowel.

2. The virtue of saline solution in the treatment of patients with obstruction in the upper part of the intestine does not lie in any protective or detoxifying influence, but in its value in replacing the chlorides and fluid lost.

3. Evidence was not obtained to show that dogs with simple obstruction in the upper part of the intestine died as the result of absorption of toxins from the obstructed bowel.

4. Interference with the continuity of the upper part of the intestine (occlusions or complete external fistula) gives rise to an increased nonprotein nitrogen in the blood and low blood chloride values; an increase in the excretion of nitrogen in the urine also occurs. The administration of sodium chloride prevents the increase in the nonprotein nitrogen of the blood, but the urinary excretion of nitrogen continues to be high.

PROBABLE INFLUENCE OF PANCREATIC JUICE IN THE REGULATION OF GASTRIC ACIDITY *

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Drainage of the total external secretion of the pancreas, as already reported in a previous communication,[1] leads to increasingly severe vomiting in a few days, and, in about a week, to a fatal outcome. In the attempt to explain these symptoms of gastric irritability, a number of observations were made. The contents of the stomach were examined in most of the animals, and the behavior of gastric acidity was studied in a special way. The results will be presented in detail. They have suggested among other things that a reciprocal relationship exists between the alkaline pancreatic juice and the acid secretion of the stomach.

One aspect of this relationship has long been known, and the data herein contained adds further proof, that is, that the acid gastric juice, passed into the duodenum, is the normal stimulus of pancreatic secretion. Its discovery by Dolinsky [2] in Pavlov's laboratory has been amply confirmed by all subsequent observers. Others of Pavlov's pupils, notably Boldyreff,[3] brought forth evidence to support the idea that by reflux into the stomach the alkaline pancreatic juice neutralized and thereby regulated the level of gastric acidity. The latter hypothesis has remained more or less unconfirmed, though in the past few years much evidence in its support has accumulated. The observations herein recorded, as will be pointed out subsequently, not only seem to support this theory, but also tend to indicate that it may be an important phenomenon.

METHODS

All observations were made on healthy adult dogs in which the total external secretion of the pancreas was being drained to the outside in a closed collecting system under aseptic conditions. This was possible by intubating the large pancreatic duct according to a method previously described.[1] In some dogs, provision was made for the return of the juice to the duodenum so that the secretion could be collected or reverted to the animal at will. The experiments here described were always performed during the first days of drainage and, therefore, before the onset of the severe vomiting.

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* From the Department of Surgery, Washington University School of Medicine and Barnes Hospital.

1. Elman, R., and McCaughan, J. M.: J. Exper. Med. **45**:561, 1927.
2. Dolinsky, J. L.: Diss., St. Petersburg, 1894.
3. Boldyreff, W.: Ergebn. d. Physiol. **11**:121, 1911.

The contents of the stomach were obtained with a small rubber tube connected at one end with a small perforated metal bulb such as is used for duodenal drainage. The animal was placed in a Pavlov frame while aspirations were performed. The acidity of the contents of the stomach were determined by titration with tenth normal sodium hydroxide with dimethyl-amino-azobenzene (Toepfer's reagent) as the end point for "free", and phenolphthalein for "total", acid. All samples were centrifugalized, since most of them contained a good deal of debris, particularly those removed from the fasting stomach. Only the clear fluid thus obtained was used in titrations.

To study the behavior of gastric acidity, a "test meal" of 200 cc. of 0.5 per cent hydrochloric acid was used as originally devised by Boldyreff.[3] The solution was deliberately introduced by gavage, and specimens were aspirated every ten or fifteen minutes thereafter till the stomach was empty. Animals were deprived of food for from four to six hours, as a rule, and the contents of the stomach were emptied before the experiment was begun.

These experiments afforded opportunity, at the same time, to measure the effect of gastric acidity on the secretion of the pancreas. The collecting tube draining the gland was connected with a sterile graduate, and the amounts of fluid flowing were recorded at regular intervals, both before and after the acid was given.

EXPERIMENTAL OBSERVATIONS

The Acid Stimulus to the Flow of Pancreatic Juice.—Fractional readings of the secretion of pancreatic juice following the introduction of 0.5 per cent hydrochloric acid into the stomach revealed an immediate and marked augmentation in the rate of flow. In the instance represented in chart 1, typical of a number of identical experiments, 51 cc. of clear sterile secretion was collected during the course of one hour after the acid was given, or nearly three times the hourly average of the entire twenty-four hour output. Though this confirms the observations of other workers, I believe that my experiments are the first performed on unanesthetized animals yielding sterile juice from the entire pancreas and must, therefore, more closely represent the normal conditions.

Failure of Acid Neutralization during Drainage of Pancreatic Juice.—Normally, acid solutions introduced into the stomachs of healthy dogs,[4] as well as healthy human beings,[5] are promptly and completely neutralized. This progressive lowering of acidity did not occur in animals in which the total pancreatic juice was flowing to the outside. It remained high, so that aside from a slight drop at first, the titrated acidity (against phenolphthalein) of the final sample was nearly the same as that of the first one. Twelve experiments were performed on eight different animals, and the results were the same. The curves in chart 2 represent an instance in a dog provided with an "altercursive" or double intubation which enables juice to drain to the outside or to

4. Boldyreff, W.: Quart. J. Exper. Physiol. **8**:1, 1914.

5. Boldyreff (footnote 4). Apperly, F. L., and Cameron, G.: Med. J. Australia **1**:521, 1923.

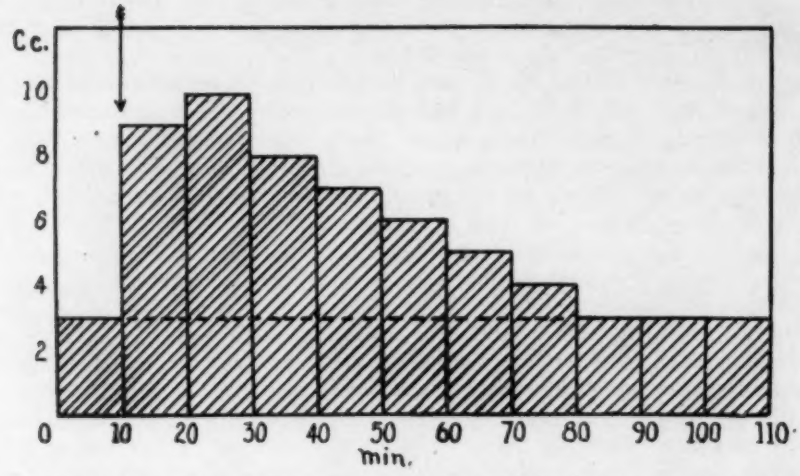

Chart 1.—The acid stimulus to the flow of pancreatic juice is shown by the prompt increase in the rate of secretion from an intubated dog following the giving of 200 cc. of 0.5 per cent hydrochloric acid by gavage (indicated by the arrow). The normal rate of flow of 3 cc. for each ten minutes increased to 9 and then to 10 cc., and gradually dropped back to the normal rate after eighty minutes. The dog weighed 10 Kg.

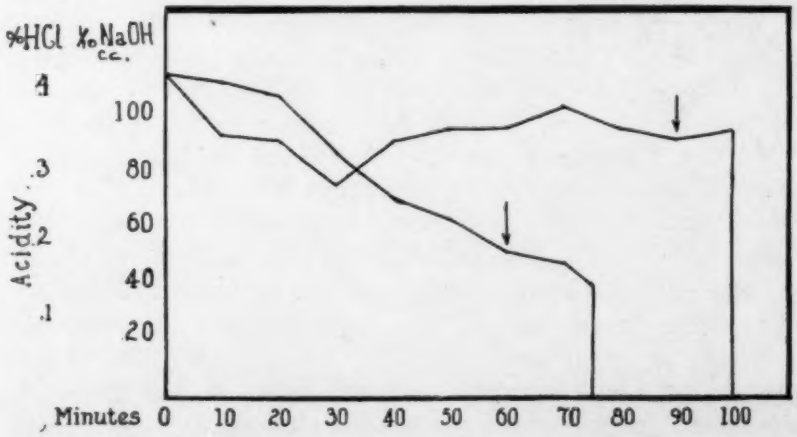

Chart 2.—The failure of acid neutralization in the stomach after the giving of 200 cc. of 0.5 per cent hydrochloric acid by gavage to a dog draining the entire pancreatic juice is shown in the upper curve. The titrations for "total" acid in successive aspirations remained high even after one and one-half hours. By contrast the prompt neutralization is shown in the lower curve of the same experiment in the same dog, but at a time when pancreatic juice was flowing back to the duodenum. The difference of twenty-five minutes in emptying time should also be noted (see text). The lower arrow indicates pancreatic juice flowing to the duodenum; the other arrow indicates pancreatic juice flowing to the outside.

flow back to the duodenum at will.[1] The lower curve shows the normal drop in the acidity of the solution introduced into the stomach at a time when pancreatic juice was flowing back to the duodenum; the upper one, the absence of neutralization when it was draining to the outside.

The experiments of Bolyreff[5] and of Migay[6] may well be referred to at this point. These authors did not find any lowering in the acidity of hydrochloric acid solutions given dogs after ligation of the pancreatic ducts or after occlusion of the pylorus. When the common bile duct or lower end of the esophagus was obstructed, neutralization proceeded normally. They concluded from this that neither bile nor swallowed saliva are responsible for the phenomenon, but rather the reflux of pancreatic juice into the stomach.

It is of special interest to note the behavior of "free" and of "combined" acid in these experiments, as shown in chart 3, which represents a typical instance. Though the former value fell, the latter rose *pari passu*, so that the "total" acid remained the same. In the normal, combined acid was not found,

Titrations of the Clear Fluid Obtained After Centrifugalization of the Contents of a Stomach Removed from Fasting Dogs Draining Pancreatic Juice Showing High Combined and Total Acid in Contrast to Those Removed from Dogs in Which the Juice was Flowing Back to the Duodenum

Dog	Hours After Food	Day of Drainage	Free Acid	Combined Acid	Total Acid
51	8	4	40	108	148
54	9	2	24	88	112
63	6	1	38	90	128
63	36	3	0	78	78
50	4	2	18	64	82
53	6	2	10	86	96
52	18	2	0	40	40
59	0	"Altercursive" fistula with pancreatic juice flowing back to the duodenum	36	16	52
59	36		28	8	36
53	4		32	12	44

as the total and free acid were always the same, the fall in acidity affecting each equally. This phenomenon may well be explained by the probable buffer action of the large amounts of intestinal contents regurgitated into the stomachs of these dogs which lose their total pancreatic juice. The samples in the normal, on the contrary, were always clear. The significance of this observation will be discussed.

The High Acidity of the Fasting Contents of Dogs Draining the Total Juice.—The contents of the stomach obtained from dogs after one or more days of drainage showed a much higher "total" acid than normal; in a few cases, it was three times as great (table). These specimens, too, showed a high value for the "combined" acid and contained a considerable amount of intestinal contents, as already mentioned.

The Regurgitation of Intestinal Contents into the Stomach.—In every instance in which the contents of the stomach were aspirated during the

6. Migay, F., quoted by Babkin, B. P.: Diss., St. Petersburg, 1909; Die äussere Sekretion der Verdaungstruse, 1914, p. 382.

drainage of the total pancreatic juice, a good deal of admixed material was found. When drainage had occurred for three or four days, the specimen was thick and brownish, and by its fecal odor it betrayed its origin in the lower ileum. Autopsy in each case showed an intact gastro-intestinal tract without evidence of gastrocolic or ileogastric fistula. It was obvious that the material in the stomach was regurgitated intestinal contents, such as are never found in the stomach under normal conditions. Centrifugalization always yielded three layers. The upper layer was grayish and proved to be neutral fat; the lower one was dark brown or black; its nature was undetermined, and there was a more or less clear fluid between the two. Titration of this fluid always showed a high content of acid.

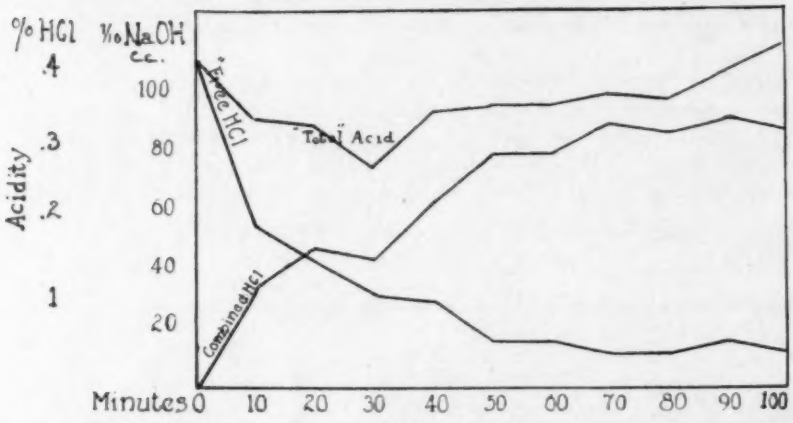

Chart 3.—The behavior of "free" and "combined" acid in titration of aspirated samples after the giving of 200 cc. of 0.5 per cent of hydrochloric acid solution to a dog draining the total pancreatic juice. After the free acid decreased, the combined acid increased so that the "total" acid was always the same. The aspirated samples always contained much regurgitated intestinal contents which probably accounted for this (see text).

COMMENT

The observations herein recorded throw considerable light on the nature of the severe gastro-intestinal upset which follows drainage of the total pancreatic secretion. In brief, they show that the contents of the stomach of such animals are abnormally acid, and, moreover, that there is a failure of neutralization to occur when acid solutions are introduced into their stomachs, though an intense flow of pancreatic juice is always provoked thereby. Finally, the constant finding of intestinal contents in the stomach of these animals shows that regurgitation occurred regularly.

The first inference from these observations was that I was dealing with a condition whereby some mechanism for the regulation of gastric

acidity was interfered with, and the second one, that the alkaline pancreatic juice was responsible. The intense regurgitation of intestinal contents into the stomach, contents even from the lower part of the ileum, pointed to an attempt on the part of the body to compensate for the missing pancreatic juice; indeed, by its buffer action, it did exert some neutralizing effect (chart 3). Analysis of the evidence of others, finally seemed also to point to the existence of this mechanism and to its importance in the relationship between stomach and duodenum, both normally and in certain pathologic conditions.

That the level of gastric acidity is actually regulated by some neutralizing agent rather than by variations in the secretory activity of the gastric glands is based on the fact that pure gastric juice is always secreted at a much higher acidity than is ordinarily found in the contents of the stomach even after a test meal. Pavlov was the first to maintain, on the basis of his own observations, that the gastric glands secrete a juice of a constant and rather high acidity of about 0.5 per cent hydrochloric acid, or, in terms of titration values, of 140.[7] More recently, Carlson,[8] though questioning the truth of Pavlov's contention, in observing two patients with esophageal stricture and gastric fistula, found that the actively secreted juice is always of a constant acidity close to 0.5 per cent hydrochloric acid. Rehfuss and Hawk reported evidence to show that human beings secrete gastric juice of a constant and high acidity,[9] and Hollander,[10] by experimentation on dogs, has shown that the actively flowing secretion from a Pavlov pouch is of a high acidity which after the injection of histamine equals a p_H of 0.92, or greater than 0.5 per cent hydrochloric acid. The much lower figures obtained in normal stomach contents must obviously be due to the existence of some neutralizing mechanism. Such a mechanism, furthermore, has been strikingly demonstrated by Kahn and Yaure.[11] These authors, using p_H as a measure of acidity, gave test meals to dogs with a Pavlov pouch and simultaneously studied the contents of the main stomach as well as the pure juice flowing from the isolated pouch. Whereas the former varied considerably and never was more acid than p_H 1.9, the pure juice was secreted at the high and constant acidity of p_H 1.1.

This regulation may occur, of course, by the diluting action of swallowed saliva, mucus, bile or pyloric secretions, and this mechanism

7. Pavlov, J. P.: The work of the Digestive Glands, London, C. Griffin & Company, 1910, p. 32.

8. Carlson, A. J.: Am. J. Physiol. **33**:248, 1915.

9. Rehfuss, M. E., and Hawk, P. B.: Gastro-Intestinal Studies, J. A. M. A. **63**:2088 (Dec. 12) 1914.

10. Hollander, F.: Proc. Am. Soc. Biol. Chem., J. Biol. Chem. **74**:23, 1927.

11. Kahn, J., and Yaure, G.: Arch. f. d. ges. Physiol. **206**:119, 1924.

has been assumed by many to account for the facts already mentioned.[12] Dilution alone must be an ineffective neutralizing mechanism, since these fluids are themselves either neutral or barely alkaline.[4] For example, to reduce the acidity of gastric juice from 140 to 35, it would be necessary to add three times its volume of a neutral fluid, whereas only three fourths its volume of pancreatic juice would be required, since it can completely neutralize more than its own volume of tenth normal hydrochloric acid. That the pancreatic juice is actually responsible for this regulation is made to appear more likely by the results of the experiments recorded in this article. All the foregoing factors have remained intact in them. By mere elimination of the external pancreatic secretion, therefore, neutralization in the stomach could not occur, and the contents of the stomach were constantly more acid than normal.

One finds frequent references in the literature to the rôle of bile in the neutralization of gastric acidity, the erroneous assumption being that bile is alkaline in reaction. All investigations have shown that hepatic bile is neutral. According to Drury, McMaster and Rous [13] the influence of the gallbladder is such as actually to make the bile acid before it arrives at the intestine (p_H from 5.1 to 6.8). It must be unlikely, therefore, that bile plays any part in the neutralization of gastric acidity.

Duodenal antiperistalsis, of course, must take place before pancreatic juice can enter the stomach. Roentgenologists have apparently rarely observed it,[14] though such evidence is merely negative. Recently, Bolton and Salmond [15] examined 100 persons, including many normal persons, with this point particularly in mind. They describe four different types of peristaltic movements in the duodenum. One of them consists of a contraction in the proximal duodenum which propels the opaque medium toward the pylorus, which, by its relaxation, permits the passage of the medium into the stomach. Such a relaxation with visible reflux was actually seen but six times, though the duodenal antiperistalsis initiating it was seen in 93 per cent of the cases.

The frequent finding of bile in the stomach can be explained only by duodenal reflux. It is not found constantly, it is true; it is known, however, that bile is not continuously discharged into the duodenum, but is dependent on periodic emptying of the gallbladder.[16] In general,

12. Ortner, A.: Arch. f. d. ges. Physiol. **186**:124, 1917.

13. Drury, D. R.; McMaster, P. D., and Rous, P.: J. Exper. Med. **39**: 403, 1924.

14. Eisen, P.: Radiol. **4**:388, 1923.

15. Bolton, C., and Salmond, R. W. A.: Lancet **1**:1230, 1927.

16. McMaster, P. D., and Elman, R.: J. Exper. Med. **44**:173, 1926. Copher, G. H., and Kodama, S.: Regulation of Flow of Bile and Pancreatic Juice in Duodenum, Arch. Int. Med. **38**:647 (Nov.) 1926.

reverse peristalsis is not uncommon in the small intestine, as pointed out so extensively by Alvarez,[17] and its infrequent visualization in the duodenum may be due to the fact that it is not being looked for and to the fact that the duodenum turns posteriorly, thus making it difficult to be seen with the fluoroscope in its entire course.

Direct evidence that pancreatic juice is regurgitated into the stomach would be furnished by the finding of pancreatic ferments in the gastric contents. This has indeed been the case. Boas[18] apparently was the first to make such examinations; he found the pancreatic ferments in a great many persons, including those who were normal. Trypsin, the most characteristic enzyme, present only in the pancreatic juice, was recovered from healthy stomachs nearly twenty years ago by a great many observers, particularly after the giving of a test meal of olive oil.[19] More recently, Rehfuss and Hawk[20] found it constantly in the fasting contents of the stomachs of normal persons. Iwanow,[21] in extensive observations on human beings, amply confirmed their observations. That trypsin may not be found when the contents are acid is due to the fact that it is destroyed in an acid medium.[22] Negative tests, therefore, are not conclusive. The frequent demonstration of trypsin in the stomach, on the other hand, can mean only that reflux of pancreatic juice has occurred.

The clinical applications of these observations chiefly concern the meaning of variations in gastric acidity found in real or supposed disease of the gastro-intestinal tract. The hyperacidity frequently found in cases of peptic ulcer, for example, has been ascribed to hypersecretion and the absence of, or low values for, acid in certain cases of so-called achylia to hyposecretion. From the evidence herein reported, it would seem that variations in the degree of regurgitation of pancreatic juice into the stomach may well be the important factor. A diminished reflux would permit the acidity to mount and account for the finding of hyperacid contents. An increased reflux, on the other hand, would so rapidly neutralize the acidity produced after a test meal that a low acidity or absence of acidity would be found. Indeed, in a recent

17. Alvarez, W. C.: The Mechanics of the Digestive Tract, New York, Paul B. Hoeber, 1922.

18. Boas, J.: Ztschr. f. klin. Med. **17**:155, 1890.

19. Mahlenbrey, J.: Zentralbl. f. d. ges. Physiol. u. Path. d. Stoffwechs. **4**: 643, 1909.

20. Rehfuss, M. E., and Hawk, P. B.: Gastric Analysis: Interdigestive Phase or Principle Governing the Resting Stomach, J. A. M. A. **76**: 564 (Feb. 26) 1921. Spencer, W. H.; Meyer, G. P.; Rehfuss, M. E., and Hawk, P. B.: Am. J. Physiol. **39**:459, 1916.

21. Iwanow, W.: Arch. f. Verdauungskr. **38**:223, 1926.

22. Kuda, T.: Biochem. Ztschr. **15**: 473, 1909. Deutsch, G., and Rurup, H.: Deutsches Arch. f. klin. Med. **138**:165, 1922.

study of achylia gastrica, Kern and his co-workers [23] found trypsin in the gastric contents in every case for which it was tested. Wright and Medes [24] reported similar observations.

The rôle of the pyloric tone in determining these variations may well be the determining factor. According to the fluoroscopic observations of Bolton and Salmond,[15] the relaxation of the pylorus at the moment of a duodenal contraction allows the immediate reflux of duodenal contents into the stomach. Confirmatory of this idea, Olch [25] found that the enlarging of the pyloric opening in dogs by pyloroplasty, results in a marked acceleration in neutralization as well as in emptying time.

From the surgical point of view, the observations herein presented cast some light on the mechanism by which various operative procedures may benefit patients with gastric and duodenal ulcers. It has been noted by many observers that the relief of symptoms usually occurs in those patients who after operation persistently show a low or absent gastric acidity.[26] In 1909, Wilcox [27] showed that the gastric contents following simple gastro-enterostomy were far less acid than before and contained bile constantly. In a recent review of all the literature, Bohmansson [28] recorded a similar occurrence in all subsequent reports. He added, however, that this anacidity tends to disappear in the course of months or years, which may be the reason why many such patients are said to suffer from a recurrence of symptoms. In his analysis of cases of gastric resection, on the other hand, he found an almost complete and permanent disappearance of acid in the gastric contents after operation. This has been the experience of many surgeons in this country as well.[29]

On the basis of the foregoing theory of regulation of gastric acidity, these results are easy to explain. After gastro-enterostomy, duodenal contents, including pancreatic juice, have an additional stoma through which to enter the stomach, thereby promoting the effectiveness of neutralization. Should the new opening close, as has long been shown to occur, the conditions would return to their former state unless the ulcer had healed in the meantime. After gastric resection, on the other

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23. Kern, R. A.; Rose, E., and Austin, J. H.: J. Clin. Investigation **11**: 545, 1926.

24. Wright, C. B., and Medes, G.: Proc. Soc. Exper. Biol. & Med. **24**: 904, 1927.

25. Olch, I. Y.: Arch. Surg., to be published.

26. Lewisohn, R., and Guinzburg, L.: Surg. Gynec. Obst. **44**:344, 1927. Lorenz, H., and Schur, H.: Arch. f. klin. Chir. **119**:239, 1922.

27. Wilcox, W. H.: Quart. J. Med. **3**:73, 1909.

28. Bohmansson, G.: Acta chir. Scandinav. **60**: 137, 1920, suppl. 7.

29. Lewisohn and Guinzburg (footnote 26, first reference). Heinbecker, Peter: Unpublished data from the Presbyterian Hospital, New York, 1926.

hand, a wide communication between the duodenum and the stomach is provided, which is the factor of importance in lowering the gastric acidity. If this is true, any type of operation which enlarges the pyloric opening would be as effective as extensive removal of the wall of the stomach. Indeed, the work of Olch [25] shows that a Finney pyloroplasty is a most effective operation in hastening gastric neutralization.

The actual clinical application of these ideas has shown that patients with proved duodenal ulcer show a marked delay in neutralization of the test meal of 300 cc. of 0.5 per cent hydrochloric acid. Following a posterior gastro-enterostomy in three of them, the "acid test meal" was rapidly neutralized, and bile was constantly present in the gastric contents. The details of these observations will be reported later.

SUMMARY

During drainage of the total pancreatic juice, the gastric contents contain a far higher total and combined acid than normal. Acid solutions introduced into the stomach of animals during such drainage provoke an intense increase in the flow of pancreatic juice to the outside, but cannot be neutralized as in the normal animal. Drainage of the pancreatic juice, moreover, is always accompanied by the regurgitation of intestinal contents into the stomach. Analysis of this and other evidence lends support to the hypothesis that reduction of gastric acidity is a normal, and probably an essential, phenomenon, which takes place through the reflux of the alkaline pancreatic juice into the stomach. Bile, being actually an acid rather than an alkaline fluid as is so often assumed, cannot play any part in this neutralization. The clinical significance of these observations has been discussed.

THIRTY-FIFTH REPORT OF PROGRESS IN ORTHOPEDIC SURGERY

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(Continued from page 1126)

MISCELLANEOUS

Orthopedic Surgery in Sweden.—Hauser [39] has given an interesting account of orthopedic developments in Sweden growing out of national, social, and health insurance legislation. Sweden early recognized that it was advisable for the community to assume the responsibility of alleviating the circumstances of the poor and needy, and a comprehensive law was passed for the benefit of all who were in need of social aid. The provisions of this law include the medical care of invalids and cripples. The system of pensioning invalids, part of the socialistic national insurance scheme which has been adopted, also influences the practice of orthopedic surgery, in that it has been found expedient to cure, or at least reduce as far as possible, invalidism before the amount of disability is fixed and a pension allocated. Under the supervision of a central committee for the care of cripples, the state organized three orthopedic centers, one at Stockholm, one at Helsingfors and one at Gothenburg. Each center comprises several units besides the diagnostic and hospital sections, and includes a home for crippled children, vocational training schools, special hospitals for the care of patients with tuberculosis of the bones and joints, and three seaside sanatoriums.

Summary of Literature Relating to Posture.—Schwartz [40] made a survey of 134 articles relating to posture, all of any importance that could be found in the literature. From this study he also concluded that most authors are in agreement on the following statements: 1. The

39. Hauser, E. D.: M. J. & Rec. **125**:789 (June 15) 1927.

40. Schwartz, L.: Pub. Health Rep. **42**:1219 (May 8) 1927.

biped posture of man has been evolved from the quadruped posture. 2. The body has not yet fully adopted itself to the biped posture. 3. There are many physical disadvantages to the erect posture, but they are outweighed by the physical and mental advantages resulting therefrom. 4. Good posture can be attained by having good health, taking enough exercise to keep the muscles strong and the joints supple, and continually assuming correct postures in the daily tasks. 5. Fatigue is the most frequent cause of postural deformities in the industries. 6. Continuous sitting or standing in any position is fatiguing. 7. Change of posture at the will of the worker is the remedy for industrial fatigue. 8. Industrial furniture should be so constructed as to fit the individual worker and to allow comfortable working conditions in both the sitting and the standing postures. The author's personal comments are as follows: 1. There is a lack of agreement in the various definitions of standards for good posture. 2. Heredity, type of build, balance of muscle strength and tone have not been given sufficient importance in establishing standards for posture. 3. It has not been established that faulty posture associated with certain diseases is the cause or result of these diseases. 4. There is no universally satisfactory test for physical fitness.

Loose Bodies in Joints.—In a discussion of the pathogenesis of loose bodies in joints, Platt [41] classified them as follows:

GROUP 1.—Diseased Joints: The patients have usually passed middle age and the loose bodies are multiple. The causes are osteo-arthritis and Charcot joints.

GROUP 2.—Otherwise Healthy Joints: Included in this group are the great number of patients with "osteochondritis dissecans" of Koenig. Platt discussed the various theories as to the etiology of the condition without reaching any conclusion.

GROUP 3.—Synovial Chrondromas: It is probable that these bodies are actually tumors from rests of cartilage cells contained in the synovial membrane. The bodies are usually multiple, may become detached, and may reattach themselves.

Madelung's Deformity.—Writing on the subject of Madelung's deformity, Catterina [42] set down the following conclusions: 1. Madelung's deformity is characterized by an abnormal curve of the radius, followed by a subluxation, more or less marked, of the ulna, and is a disease that occurs in adolescence, especially in girls. 2. Such lesions must be attributed to rickets. Vigorous movements, as of dorsal flexion of the hand due to special occupations, may accentuate the pre-

41. Platt, Harry: Brit. M. J. **1**:947 (May 28) 1927.

42. Catterina, A.: Chir. d. org. di movimento **10**:517 (April) 1926.

existing radial curvature and induce other skeletal lesions. 3. Orthopedic treatment gives best results if initiated early, and surgical treatment should be attempted only in the severe cases and consists of oblique linear osteotomy of the inferior third of the radius.

Commonly Confused Disabilities of the Hip Joint in Children.—As a basis for study Dillehunt [43] made an analysis of 1,000 patients consecutively admitted to the Shriners' Hospital for Crippled Children and the Orthopedic Clinic of the University of Oregon Medical School. He found that of this number ninety-four, or nearly 10 per cent, were children, under the age of 16 years, with disabilities of the hip joint. Of the ninety-four patients, twenty had tuberculosis, fourteen Legg's disease, six coxa vara, twenty-eight congenital dislocation, fourteen osteomyelitis or septic arthritis, and six chronic arthritis of unknown cause. Discussing the diagnosis of infections of the hip, Dillehunt made the following points: 1. If a joint cannot be moved in any direction without severe pain, generalized disease is present within the joint. 2. If the motion of the hip is painful when performed in certain directions, but is painless in one or more other directions, the disease is close to, but outside, the joint. 3. Whenever osteomyelitis occurs in a bone near a joint, there occurs in that joint a synovial effusion containing many leukocytes, which looks like pus, but on aspiration and culture is found to be sterile. To drain such a joint assures its destruction and neglects the real disease, the osteomyelitis. 4. It is always safe to aspirate a joint under aseptic precautions, and if the fluid withdrawn is clear or slightly blood-tinged, and culture reveals the streptococcus, then the best immediate treatment is to immobilize it with traction and not to drain it. If the fluid is frank pus containing staphylococci, the joint should be drained widely. No matter what the fluid looks like, if it is sterile it should not be drained.

Pulmonary Embolism Following the Injection of Oxygen into the Knee.—Kleinberg [44] reported the case of a patient with disability of the knee in whom he performed insufflation of the joint for roentgenodiagnostic purposes. The injection of the oxygen through the needle had scarcely begun when the patient collapsed, with all the symptoms of pulmonary embolism. The patient appeared to be dead, but he was revived by the intracardiac injection of epinephrine hydrochloride and eventually recovered. Kleinberg supposes that the needle happened to be inserted in a vein and that the case was one of true pulmonary embolism, the bubble of gas being expelled from the lungs when the heart action recommenced.

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43. Dillehunt, R. B.: Northwest Med. **26**:254 (May) 1927.

44. Kleinberg, Samuel: Pulmonary Embolism Following Oxygen Injection of Knee, J. A. M. A. **89**:172 (July 16) 1927.

[ED. NOTE.—The danger of pulmonary embolism while injecting air or gas into the knee joint apparently is not great, but it is evidently something to be guarded against by careful precaution.]

Etiology of Lesions of Semilunar Cartilages.—Galeazzi [45] concluded that the general accepted view of the method of production of traumatic lesions of the semilunar cartilages is obscured by much theoretical speculation. According to the generally accepted views, lesions of the menisci are caused by the action of rotary forces working on either the tibia or the femur with the knee in partial flexion. Anatomic investigations have demonstrated the existence of intimate ligamentous connections between the cartilages and the crucial ligaments. Experimentally, the author succeeded in producing lesions of the menisci of the cadaver. In patients he frequently found these lesions associated with partial or complete rupture of the crucial ligaments. He concluded that they are to be accounted for as resulting from excessive tension of the crucial ligaments transmitted to the menisci, or, in other words, that an indirect trauma is transmitted through the crucial ligaments.

Bilateral Relaxation of the Superior Tibiofibular Articulation.—Bilateral relaxation of the superior tibiofibular articulation causing symptoms is a rare condition, but Schoolfield [46] saw and recorded such a case. The condition was bilateral, but only on one side did it give rise to symptoms. The author performed an arthrodesis between the two bones which resulted in complete relief.

Coalition of Calcaneus and Navicular.—Badgley [47] encountered during the past year several patients with coalition between the calcaneus and the navicular bone of the foot. The extent of the coalition varied from fusion between the calcaneus secondarius and the medial end of the calcaneus in different degrees, to the formation of a complete bony bridge uniting the calcaneus and the navicular. In all cases there was severe disability due to rigid flat foot. The rigidity of the foot was in proportion to the amount of fusion. The author considers the condition fairly common and ascribes his own failure previously to recognize it as due to the fact that the roentgenograms had not been made in the proper oblique position. Removal of the bony bridge may restore mobile function in certain cases, but when the condition is in an advanced stage, arthrodesis of the substragalar, calcaneonavicular, and calcaneocuboid joints may be required.

Disease of the Sesamoid Bones of the First Metatarsophalangeal Joint.—To the four cases of disease of the sesamoid bones of the first

45. Galeazzi, R.: J. Bone & Joint Surg. **9**:515 (July) 1927.

46. Schoolfield, B. L.: J. Bone & Joint Surg. **9**:500 (July) 1927.

47. Badgley, C. E.: Coalition of Calcaneus and Navicular, Arch. Surg. **15**:75 (July) 1927.

metatarsophalangeal joint which Mueller [48] previously described he added the report of three more. The clinical signs which he noticed were pain, tenderness and moderate swelling under the affected area. Roentgen-ray examination showed the medial sesamoid bone divided in from two to three fragments. The patients were from 20 to 30 years old. The cause, according to the author, was occupational strain. Histologic examination revealed necrotic areas in the bony tissue but not in the marrow. Mechanical irritation is supposed to have resulted in fragmentation of the necrotic bone. The treatment is stated to be support of the foot in patients with slight symptoms and operative excision when the symptoms are severe.

BONE, JOINT, AND TENDON SURGERY

Spinal Anesthesia in Orthopedic Surgery.—Peabody [49] employed spinal anesthesia in 100 orthopedic operations and feels that it is particularly adapted to this class of cases. He found that it may be applied successfully in all procedures below the level of the dorsal spine, and his experience supplements that of others in establishing that it is a safe form of anesthesia.

Pneumatic Hammer for Operation on the Bone.—Pitkin [50] described a pneumatic hammer for use in bone surgery. It is an adaptation of the Boyer *U*-type hammer manufactured by the Chicago Pneumatic Tool Company. The author described this hammer as a simple tool, sturdy, easily cared for, and safe.

A Pendulating Bone Saw.—Not satisfied with the commonly used bone saws, Von Baeyer [51] constructed a pendulating saw. The ordinary rotary electric saw cannot be used in deep incisions unless a large blade is attached, and this represents a decided danger for the soft tissues. The author's saw is constructed with two swinging saw blades which work in different directions. They are driven in opposite directions with great rapidity and slight vibration by an electric motor. It is stated that an average sized long bone can be cut through in thirty seconds with this instrument.

Operative Splinting of the Vertebral Column in Pott's Disease.—Lange [52] reported his experience with fifty-two patients with Pott's disease in whom he performed operative splinting of the spine by introducing celluloid splints of from 5 to 10 mm. in diameter and from 10 to 30 cm. in length. As he described it, his technic is to expose the spinous

48. Mueller, W.: Beitr. z. klin. Chir. **138**:494, 1926.
49. Peabody, C. W.: J. Bone & Joint Surg. **9**:450 (July) 1927.
50. Pitkin, H. C.: J. Bone & Joint Surg. **9**:505 (July) 1927.
51. Von Baeyer, H.: Zentralbl. f. Chir. **54**:2394, 1927.
52. Lange, Fritz: Surg. Gynec. Obst. **44**:668 (May) 1927.

processes and separate the muscles from their sides down to the vertebral arches. "Each of the exposed spinous processes is perforated at the upper half and a loop of silk thread (no. 12) is carried through the hole. . . . Through a second hole, drilled a little below or through the interspinous ligaments, the same loop of thread is returned to the other side, leaving thus a *U*-shaped loop of double thread on one side and the four open ends on the other. A celluloid splint which has been fitted exactly to the curve of the gibbus is inserted in the loop and another similar splint is placed on the other side of the spinous processes. The latter splint now being grasped between the free ends of the thread with a firm tension is securely tied to the spinous processes with a knot." The splints are fixed in similar fashion to each of the spinous processes. "They are then carefully covered up with muscles and fascia and sutured in the ordinary way with silk no. 6." A plaster of paris dressing is used for six weeks and then a corset is worn for two years. Since 1924 he has used one splint of rust-proof steel and one of celluloid. In reference to Albee's and Henle's modifications Lange said that he preferred his own method, as it involved less danger of fracture of the splint, and the bone splint requires a prolonged dressing with plaster of paris and a stay in bed of six months or more.

[ED. NOTE.—Either of the methods commonly employed for obtaining bony fusion of the spine, Albee's or Hibbs', seems so far superior to Lange's method of securing fixation by means of celluloid splints that we consider it unnecessary to point out the advantages of the former or the disadvantages of the latter.]

Transplantation of Trapezius Muscle for Paralysis of the Abductors of the Shoulder.—Mayer [53] described a method of transplantation of the trapezius muscle for paralysis of the abductors of the shoulder. It consists, briefly, in detaching the trapezius muscle from its bony insertion, lengthening it by means of an artificial tendon constructed of fascia lata, and suturing this tendon to the humerus near the deltoid insertion. To secure a satisfactory result it is necessary that the trapezius, the serratus magnus, and the pectoralis major, or the coracobrachialis or the biceps, be present and active. The author reported the results in six patients: one failure, one poor result, and four gratifying results.

Habitual Dislocation of the Shoulder.—Still another operative procedure has been added by Carrell [54] to the long list of those previously described for the relief of habitual dislocation of the shoulder. The author's operation is stated to combine the principles of the suspension and reefing procedures. The suspension element is accomplished

53. Mayer, L.: J. Bone & Joint Surg. **9**:412 (July) 1927.

54. Carrell, W. B.: Habitual Dislocation of Shoulder, J.A.M.A. **89**:948 (Sept. 17) 1927.

by using the tendon of the biceps, to which is fastened a long strip of fascia. This is then passed under the neck by a special instrument, penetrating the capsule in two places, and is secured in a drill hole in the acromion. This gives a slinglike action and anchors the head in the glenoid as well as to the acromion. The reefing feature has the advantage of plicating a heavy fascial band through the weak portion of the capsule, and the resulting fibrosis should leave a thick band encircling the neck and continuous with the suspension band. The operation was performed on four patients, and unsatisfactory results have not yet been seen. Ill effects from changing the biceps origin have not been noted.

[ED. NOTE.—It seems to us that the operation described does not offer any particular advantage over the method of direct fixation of the humeral head to the acromion by a fascial band. It appears considerably more complicated.]

Operations on the Upper Extremity.—Steindler [55] analyzed the kinetics of the upper extremity and the various operative measures employed for the relief of paralysis of the shoulder, of the elbow, and of the wrist. Arthrodesis of the shoulder was performed in sixty-five patients because of paralysis of the deltoid. On the whole, the results were satisfactory, but not uniformly so. He found that the optimum functional position varied in children and adults; in children, 90 degrees of abduction, or rather forward flexion, is favored; in adults, from 60 to 70 degrees. The author's operation for relief of paralysis of the flexor muscles of the elbow was performed on thirty patients, and the results have been satisfactory. The operation consisted of transplanting the common origin of the pronator teres, flexor carpi ulnaris, palmaris longus, and flexor carpi radialis muscles to a point higher up on the humerus. Forty-two operations were performed for relief of pronation contracture, mostly of the spastic type. The operation consisted of a resection of the pronator teres or pronator quadratus, and tendon transplantation of the pronator and flexor carpi ulnaris to the dorsum of the forearm. Paralyses of the wrist were treated by the operation of arthrodesis in fifty-four patients, and by tendon transplantation in thirty-four patients.

[ED. NOTE.—Steindler is a careful student and has had a great deal of experience in the treatment of paralytic conditions of the upper extremity which entitles his opinion to respect.]

Loop Operation for Paralysis of Adductors of Thumb.—A loop operation for relief of paralysis of the adductor muscles of the thumb has been originated by Mayer.[56] This operation retains the essential features of the so-called pulley operation of Bunnell. It consists of passing

55. Steindler, A.: J. Bone & Joint Surg. **9**:404 (July) 1927.
56. Mayer, L.: Am. J. Surg. **2**:456 (May) 1927.

the sublimus tendon of the ring finger through a loop formed by uniting the two ends of the free transplant of the sublimus tendon of the middle finger. The free end of the sublimus tendon of the ring finger is carried diagonally across the palm and drawn through a drill hole in the base of the proximal phalanx of the thumb. In the five patients on whom the operation was performed, there is excellent opposing action of the thumb and the usefulness of the hand has been much improved. The transplanted tendon can be felt to glide freely through the pulley loop.

Reconstruction of the Crucial Ligaments of the Knee.—Horan [57] reported the results of reconstruction of the anterior crucial and internal lateral ligaments of the knee in eleven patients operated on by Alwyn Smith. The latter's modification of Hey Groves' operation (fascial substitution) was employed. All the patients were pensioners, and for this reason their responses were somewhat cautious, but with three exceptions good knees were reported. In one patient an arthrodesis was performed five years after the original operation, and during this procedure a strong viable crucial ligament was found. The author concluded that the operation results in a joint capable of withstanding the stress and strain to which it is subjected in the course of an average life.

Arthrodesis of the Ankle.—For the condition of complete paralysis of the foot Straub [58] advocates the operation of pseudarthrosis of the ankle, including the tibio-astragalar, the subastragalar, the calcaneo-cuboid, and the astragaloscaphoid joints. To make the astragalus completely fill the space between the malleoli he splits the astragalus in the mid-sagittal plane, after removing the cartilaginous surfaces, and inserts a disk of bone cut from the anterior pole of the head of the astragalus. This separates the two halves of the astragalus and forces the outer surfaces of the halves against the inner surfaces of the malleoli.

Etiology of Claw Foot.—Royle [59] made a study of the etiology of claw foot and concluded that the primary condition is a weakness of the gastrocnemius muscle, with a compensatory increase in the function of the tibialis posterior muscle. Proceeding on this theory, he suggested that the logical treatment is transplantation of the tibialis posterior tendon into the gastrocnemius muscle. He performed this operation on nine patients, with satisfactory results.

Reproduction of a Metacarpal Bone by a Bone Graft.—Following excision of the entire fifth metacarpal bone Fowler [60] succeeded in replacing it by grafting an autogenous transplant from the tibia. The

57. Horan, Maurice: Brit. J. Surg. **14**:569 (April) 1927.
58. Straub, G. F.: Surg. Gynec. Obst. **44**:675 (May) 1927.
59. Royle, N. D.: J. Bone & Joint Surg. **9**:465 (July) 1927.
60. Fowler, Andrew: Brit. J. Surg. **14**:675 (April) 1927.

distal end of the graft was rounded and its proximal end implanted firmly in the carpus. Now after two and one-half years, he reports that nearly perfect function was present.

FRACTURES

Metal Aids in the Treatment of Fractures.—Rossi,[61] from a study of twenty-nine patients with fractures of the long bones, treated by internal metal fixation, drew the following conclusions: 1. In compound fractures of long bones, osteosynthesis with metallic means of direct fixation of the fragments furnishes, in many cases, a useful and necessary accompaniment either directly or after an immediate and careful cleansing of the wound. Open postoperative treatment of the wounds of compound fractures, if well immobilized, is not incompatible with the presence of metallic material. 2. When in simple fractures of long bones the roentgenograms reveal that the ordinary methods of reduction and fixation are not likely to obtain satisfactory reposition of the bones, early osteosynthesis represents the only method of treatment which is capable of giving perfect correction. Since the purpose of osteosynthesis is to secure exact temporary retention of the fragments during the period of consolidation, it must be strong enough to overcome the action of the muscles. The method of internal fixation should be simple, and capable of application with simple technic. 3. In the choice of the means of internal fixation, good judgment must be exercised. The site and accessibility of the fracture should be considered and the advisability of leaving the means of synthesis in the tissues. 4. While in compound fractures as well as in simple fractures foreign material such as metallic wire may be left in the tissues, it is necessary to remove the plates in compound fractures, and it is better to remove them when the consolidation is satisfactory in subcutaneous fractures, especially in sites like the tibia.

Nerve Injuries Complicating Colles' Fracture.—Nerve injuries complicating Colles' fracture are generally considered uncommon, but Turner [62] expressed the opinion that this is not the case. He found evidence of nerve involvement in several patients and was led to the belief that only on this basis can the so-called trophoneuroses of the hand that occasionally develop after Colles' fracture be explained. The author's views may be summarized as follows: The dorsal branch of the ulnar nerve lies over the ulnar head and often comes in close approximation to a fracture there. At the same level, or more distalward, lies the volar branch of the ulnar. The median nerve passes through the same region, but it is usually separated from the fracture site by the flexor pollicis longus and

61. Rossi, Aurelio: Chir. d. org. di Movimento **10**:486 (April) 1926.

62. Turner, H.: Arch. f. klin. Chir. **128**:422, 1924.

the pronator quadratus muscles. The volar interosseous nerve runs more deeply and is usually in no danger of injury. The radial nerve may sometimes be injured, but the dorsal interosseous nerve is least favorably situated to escape injury. It lies in direct contact with the dorsal surface of the distal end of the radius. Careful study in the first few days of each patient with Colles' fracture will, according to the author, often reveal evidence of a nerve injury. The usual signs are excessive perspiration in the palm, hyperesthesia on the palmar side of the carpal bones, hyperesthesia of the thenar eminence, and pain over the ulnar nerve distribution, particularly when the ulnar styloid has been torn off. Bruising or overstretching are considered common, but are overlooked because the attention of the surgeon is focused on the change in the bone. The most serious changes occur in injuries of the dorsal interosseous nerve. Here after a few days, sometimes only after a week or more, a hard edema appears on the dorsum of the hand, extending to the middle third of the forearm and distalward to the second phalanges of the four fingers. The skin is glossy and usually reddened. Stiffness of the fingers soon follows, with limitation of motion both actively and passively. If one attempts motion with force, there is sharp pain in the fingers and forearm. An extension contracture of the fingers develops with slight flexion at the proximal interphalangeal joints. Motion also becomes decreased at the wrist joint. Later an actual swelling of the proximal interphalangeal joints may develop. The condition remains indefinitely without any change. Roentgen-ray examination of the hand shows increased radiability and haziness of the lower end of the radius, carpal bones, and metacarpal bones. The changes are more marked in the third and fourth metacarpal bones, and less in the second and fifth, and there is not any change in the first metacarpal bone. The changes are seen to be less marked as the examination proceeds up the forearm. The essential changes are osteoporosis and a disintegration of the internal architecture of the bone. Two cases are reported in which the changes mentioned occurred. In the second case an exploratory operation was performed on the dorsal interosseous nerve. The nerve in the region of injury was found to be somewhat spindle-shaped and congested. A slight improvement followed the exploratory operation, with less edema and less stiffness of the fingers. The author concluded that this condition is a trophoneurosis produced originally by trauma and attributable to an injury of the dorsal interosseous nerve. He added as proof three more cases, in each of which the patient was bitten by a cat over the course of the dorsal interosseous nerve, and in which the same neurologic disturbances occurred as in the cases of Colles' fracture. The author did not suggest any treatment for the condition, although slight improvement did occur in one case following operation.

[Ed. Note.—The article by Turner was published in 1924, but was overlooked at that time and so not included in the Report of Progress in Orthopedic Surgery. Only recently was our attention called to it, but it appeared significant, and for this reason it is abstracted in this report. We have observed several patients who unaccountably developed painful stiff hands after Colles' fracture, and Turner's theory affords a plausible explanation.]

Late Results of Fractures of Long Bones.—Walker [63] has continued with his studies of the late results of gunshot and other fractures among the American combatants in the World War. He reviewed the results in 4,647 fractures of the femur. He compared the results in veterans who applied to the bureau for relief twelve, eighteen, twenty-four, thirty, and thirty-eight months after injury. After a period of four years, the examination showed an improvement in disability rating of 86 per cent in the twelve months group; 77 per cent in the eighteen months group; 62 per cent in the twenty-four months group; 45 per cent in the thirty months group, and only 14 per cent in the thirty-six months group.

Epiphyseal Separation of the Ischium.—Schoolfield [64] described the rare case of a patient with epiphyseal separation of the ischium. Roentgen-ray examination showed an inward displacement with considerable encroachment on the pelvic cavity, the ilium and pubes apparently remaining in normal relationship. There was a defect in the acetabulum. The head and neck of the femur showed changes characteristic of Legg-Calvé's disease. A perfect reduction was finally obtained following two manipulations and fixation of the hip in a position of wide abduction.

Fractures of the Knee.—Zanoli [65] made a study of fractures in the region of the knee, excluding fractures of the patella, using the material of the Rizzoli Institute. Between the years 1899 and 1922, thirty-four patients with such fractures were treated of a total of 1,864 fractures, thus making a proportion of 1.23 per cent. Interarticular fractures of both the lower end of the femur and the upper end of the tibia were included. From a study of the results the writer concluded that conservative treatment, using the traction apparatus of Zuppinger until replacement of the fragments is obtained and then immobilization in a plaster casing, is the method of choice.

[Ed. Note.—We do not know of any more difficult fracture problem than that represented by some of the interarticular fractures of the upper end of the tibia. The entire joint surface may be disrupted, and

63. Walker, J. B.: U. S. Vet. Bur. M. Bull. **3**:651 (July) 1927.

64. Schoolfield, B. L.: J. Bone & Joint Surg. **9**:498 (July) 1927.

65. Zanoli, R.: Chir. d. org. di Movimento **10**:463 (April) 1926.

injuries of the semilunar cartilages and of the crucial and lateral ligaments are frequent accompaniments. The only chance of restoring function in such a knee lies in early operative treatment. There is such wide variation in the damage caused by these fractures that it is impossible with fairness to group them in one class and conclude, as the author of the foregoing article has done, that nonoperative treatment is the method of choice.]

RESEARCH

Influence of Diet on Healing of Wounds.—Herrmannsdorfer,[66] who in collaboration with Sauerbruch previously studied the effects of diet on patients with surgical tuberculosis, has now gathered together his observations bearing on the effects of diet on wounds and infections. He found important differences between the metabolism of healthy persons and that of persons with infected wounds. The reaction of infected wounds is acid, and the more severe the infection the greater the acidity. The alkali reserves of the blood are lowered. He stated that it is possible by suitable diet to influence the acidity of the wound and that this can be demonstrated. With an acid diet the weight of the body is in balance, the wound shrinks, the secretion decreases, and the bacteria diminish, in both variety and number. On the other hand, with an alkaline diet the weight of the body increases and the appearance of the wound changes for the worse. The granulations swell, the wound secretion becomes greater in amount, a pseudodiphtheritic membrane spreads over the surface, and the bacteria are increased in number and in variety. The writer expressed the belief that the experiments clearly show that the living tissues are subject to great variation in chemical composition and that these changes have an influence in determining whether bacteria will find them favorable or unfavorable culture mediums.

[ED. NOTE.—We report this article with reserve, hoping that it may stimulate others to repeat the experiments and either confirm or disprove Herrmannsdorfer's observations.]

Joint Fluid Ferments.—Abderhalden and others have shown that the juices of certain organs have specific reactions resembling those of ferments in that they act only on peptones obtained from those organs. Hempel [67] tested synovial fluid on joint cartilage, using Abderhalden's microtechnic. In six of twenty-two patients specific ferments were demonstrated; in seven patients the results were negative, and in the remaining patients the joint fluid was not of a character to permit optical examination. In four of the six positive tests, the cartilage was distinctly affected. The reaction is said to be so exact that a lesion of

66. Herrmannsdorfer, A.: Deutsche Ztschr. f. Chir. **200**:534, 1927.

67. Hempel, C.: Zentralbl. f. Chir. **54**:2377, 1927.

the cartilage which is invisible macroscopically may give a positive result. Hempel also pointed out the close relationship between the blood and the joint fluid, as shown by the similarity in the contents of both fluids.

Healing in Injuries of the Carpal Scaphoid.—Johnson [68] carried out a series of experiments on animals to demonstrate the reparative processes after injury in the short cancellous aperiosteal type of bone, such as the carpal scaphoid. The same operative procedure was performed in each of a series of adult dogs. In one forepaw the scaphoid was fractured with an osteotome; in the scaphoid on the opposite side a deep defect was created with a small drill. At the same time, in the corresponding radius, an osteotomy was performed or a defect made in the lower third of the bone. The dogs were then killed at various time intervals up to six weeks. From a study of the specimens thus obtained the author drew the following conclusions: 1. Bone repair takes place in the scaphoid in exactly the same manner as in the medulla of the diaphyses, but the process is not as active or as extensive. 2. There is no interference with vascularity of the fragments which would cause necrosis, delayed union, or nonunion. 3. In these experiments approximation of fragments was so close that mobility or displacement did not play any part in retarding union. 4. Lytic action of the synovial fluid was not observed. 5. Lack of all periosteal callus is a large factor in causing slower union. 6. Fractured hyaline cartilage heals by fibrous tissue, and adjacent but undamaged hyaline cartilage also often undergoes fibrous change. This gives rise to permanent changes of the articulating surfaces and is probably the element most responsible for poor functional results seen clinically in these fractures. 7. All bones of this cancellous type healed more slowly than the long bones. This is due largely to the lack of subperiosteal callus formation, but it is also due, in part, to the cancellous reaction being less extensive and less active than is the medullary response in the diaphysis.

68. Johnson, R. W., Jr.: J. Bone & Joint Surg. **9**:482 (July) 1927.

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